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THE SURGICAL TREATMENT OF BRAIN ABSCESS BY EXPOSURE AND ENUCLEATION

ROBERT A. GROFF, M.D.

AND

FRANCIS C. GRANT, M.D.

PHILADELPHIA, PA.

FROM THE NEUROSURGICAL SERVICES OF THE HOSPITAL OF THE UNIVERSITY OF PENNSYLVANIA AND GRADUATE HOSPITAL OF THE UNIVERSITY OF PENNSYLVANIA, PHILADELPHIA, PA.

THE PROCEDURE of enucleation of an "encapsulated" brain abscess was considered, by Sargent,[1] to be the best method of treatment. He reported, in 1928, without giving details, five patients successfully treated in whom the abscess had been removed. More recently, Vincent[2] has renewed the interest in this method of treatment and has proved its value. Up to the present time, he has reported five patients. The abscess, in each instance, was exposed and removed without rupture. By our successful use of this method of treatment for brain abscess in six of seven patients, we wish to add further support to the procedure of complete enucleation, and from the experience gained, outline the surgical management of a patient in whom the diagnosis has been made.

Case 1.*—*Synopsis: Intracranial symptoms for six weeks. Signs of intracranial hypertension and bilateral sixth and seventh cranial nerve involvement. Ventriculogram: Lesion in right frontoparietal lobes. Craniotomy and complete removal without rupture of a walled abscess. Recovery.*

E. S., Hosp. No. 125122, female, age 20, was admitted to the Graduate Hospital, September 12, 1936, having been referred by Dr. J. C. Yaskin, Philadelphia, Pa. About three months prior to admission, the patient had had a tonsillectomy performed, under local anesthesia, without any untoward effects. Six weeks later she began complaining of headache and somewhat later of diplopia, loss of vision and vomiting. During the few days preceding admission to the hospital the pain in the head had become more severe.

Physical Examination.—Temperature 99.3° F., pulse 64, respirations 22. Neurologically, she was mentally clear, had some rigidity of the neck, a bilateral Kernig, a bilateral papilledema of about three to four diopters, a cut in the left temporal field, weakness of both external recti muscles, more marked on the left than on the right, a paralysis of both seventh nerves, more marked on the right than on the left, and no significant changes in the extremities. The spinal fluid pressure was 350 Mm. of water, and the spinal fluid showed five lymphocytes.

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* Previously reported as Case 1, Brain Abscess of Undetermined Etiology, J. C. Yaskin, F. C. Grant, and R. A. Groff, ANNALS OF SURGERY, **107**, 492, April, 1938.

The white blood count was 12,800; with a mild secondary anemia. The urinalysis was negative. The blood Wassermann was negative. The stools showed ova and segments of *Taenia saginata*; the patient's father recalled that she had had a tapeworm many years ago. Roentgenographic examination of the skull and the paranasal sinuses revealed no abnormalities. A second lumbar puncture revealed a pressure of 700 Mm. of water, and the spinal fluid showed 15 cells.

Operation.—September 19, 1936: Dr. F. C. Grant did a ventriculogram which showed a mass lesion on the right side, probably in the frontoparietal region. A right frontotemporal bone flap was reflected. The dura was extremely tense, and upon its reflection, the sylvian fissure was seen to be pushed up by a mass beneath the surface and within the substance of the tip of the right temporal lobe (Fig. 1). A transcortical incision was made over the tumor which was removed without rupture. It measured 3½x4x5 cm.

Pathologic Examination.—Section of the tumor revealed an abscess filled with a fibropurulent, thick exudate. The wall was 0.5 cm. thick. Microscopic examination of the

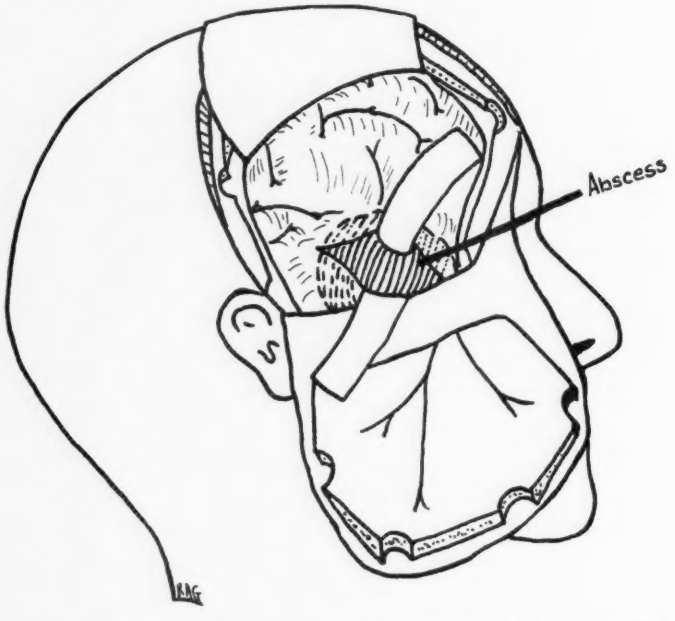

FIG. 1.—Case 1: Schematic diagram showing location of abscess in right temporal lobe.

wall showed it to be composed of a stout fibrous stroma, in which many short glial fibrils were to be seen together with numerous blood vessels, many of which showed proliferative changes. Plasma cells, lymphocytes, and old polymorphonuclear cells were present.

Subsequent Course.—The patient made a rapid and uneventful convalescence. The neurologic signs disappeared and she has remained well to date.

COMMENT.—The history given by this patient should have suggested the possibility of brain abscess. The relatively long interval between the tonsillectomy and the development of intracranial symptoms seemed to exclude this diagnosis. The bilateral sixth and seventh cranial nerve impairment, without other signs except intracranial hypertension, made ventriculography necessary. The fortunate surgical management of the lesion by intact, complete removal prevented the fatal complication—meningitis. The smooth un-

complicated convalescence and the continuance of the patient's good health up to the present time, two and one-half years, commend this method of treatment.

Case 2.—*Synopsis: Symptoms for 14 weeks. Signs suggestive of expanding left cerebral lesion. Cannula struck abscess during ventriculography. Abscess exposed and removed without rupture. Recovery.*

J. F. M., Hosp. No. 35238, male, age 26, was admitted to the University Hospital, January 12, 1937, having been referred to Dr. F. C. Grant by Dr. Marshall W. Dyer, Syracuse, N. Y. The patient was well until 14 weeks before admission to the hospital, when he began complaining of projectile vomiting. This vomiting bore no relationship to meals or time of day and continued up until the time of admission. One week after the onset he developed left-sided headache which subsequently became generalized. Shortly afterward he had periodic attacks of dimness of vision. While walking he noticed he tended to deviate to the right and on numerous occasions became dizzy when bending over.

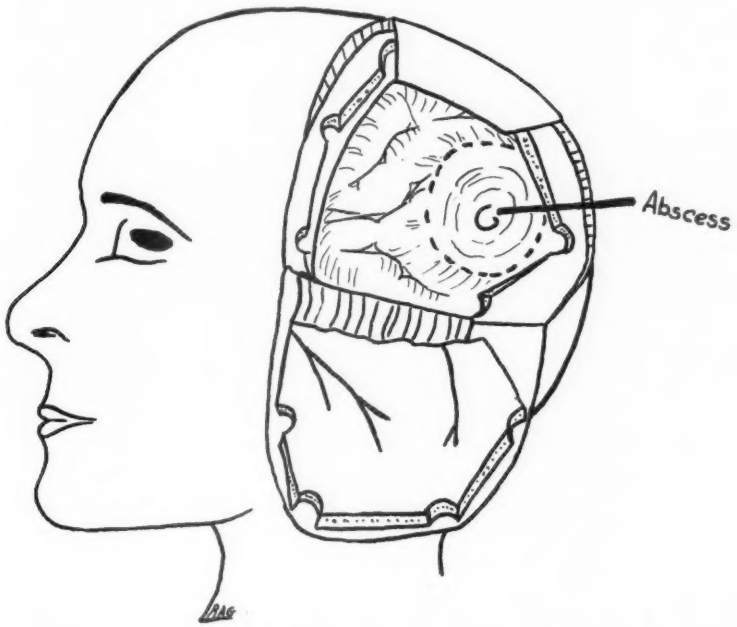

FIG. 2.—Case 2: Schematic diagram showing location of abscess in left occipital lobe.

Physical Examination.—The patient was normal except for dental and tonsillar sepsis. Neurologic examination revealed a fair mental orientation, dysarthesia with test phrases, left sixth nerve palsy, choked disks of four to five diopters, positive tremnor sign on the right, abortive ankle clonus on the right together with mild increase in reflexes on the right side of the body, dysnergia in finger to nose test on both sides, unsteady gait, and poor associated movements on the right side. Temperature, pulse and respirations were normal. The leukocyte count 10,600, and the Wassermann negative. The visual fields showed a marked contraction of both temporal fields, especially on the right, and a complete right homonomous hemianopia. Roentgenograms of the skull showed erosion of the posterior clinoids and dorsum of the sella with some forward displacement of the top of the dorsum.

Operation.—January 22, 1937: Dr. F. C. Grant performed a ventriculogram for the purpose of localization. A solid mass was encountered by the cannula when attempting to enter the posterior horn of the left lateral ventricle. Predicated upon this, a left

occipitoparietal bone flap was reflected. On the cortex, in the occipital lobe (Fig. 2), a grayish-yellow, massive tumor was exposed. This tumor was carefully dissected from its bed without damaging it. After complete hemostasis had been secured, the wound was closed. The tumor measured approximately 6x4x4 cm.

Pathologic Examination.—Section of the tumor revealed it to be an abscess containing thick greenish pus. The wall was quite thick and contained a few large blood vessels. Microscopic examination of the wall showed it to be composed of a dense fibrous structure arranged in parallel rows. Among these fibers were numerous fibroblasts and scattered polynuclear cells.

The patient made a rapid and uneventful convalescence. On discharge practically all of the neurologic signs had disappeared but the disks showed a choking of two diopters.

Subsequent Course.—The patient has been seen since operation. The vision and visual fields have improved. However, in the six months following removal of the abscess, three generalized major convulsions have occurred. He was given small amounts of phenobarbital and has had no attacks for the past 18 months.

COMMENT.—This patient presented essentially the same problem as the case just described. The history was longer and the neurologic signs were suggestive but not conclusive of exact localization. The abscess was exposed by an osteoplastic flap and completely removed without damage. The rapid convalescence and the necessity of only 20 days' hospitalization further emphasize the rationale of this method of treatment.

Case 3.—*Synopsis: Intracranial symptoms for two weeks following mild head injury and streptococcic sore throat. Signs of intracranial hypertension and a right parietal lesion. Craniotomy, tap of abscess by exploring cannula, and complete enucleation. Recovery.*

S. K., Hosp. No. 39361, male, age 21, was admitted to the University Hospital, June 6, 1938, having been referred by Dr. C. C. Neff, York, Pa. The patient had been perfectly well until two weeks before admission, when he bumped his head on a beam. He did not become unconscious nor were there any ill effects from this accident. Shortly afterward the patient developed a sore throat which lasted one week. The infection was alleged to be caused by the streptococcus. One week before admission he had an attack in which both arms and legs "stiffened," but no loss of consciousness occurred. This attack began in the left arm and spread to involve the rest of the body. It lasted approximately ten minutes. During the next three days he had three to four similar attacks. Since then the patient has become progressively more dizzy, especially when attempting to walk. At about the same time these attacks began he developed right frontal headaches. For the several days before admission the left arm and hand had become weak.

Physical Examination.—The patient was acutely ill. Temperature 99° F., pulse 80, respirations 20. Neurologic examination: The patient was definitely lethargic, the eyegrounds showed a bilateral papilledema of between two to three diopters. The visual fields, to gross tests, were normal. There was a left central facial weakness. The corneal reflex was decreased on the left. The left arm and hand were weak in all movements and a similar but less marked weakness was demonstrated in the left leg. Reflexes in the left arm were increased over those in the right. In the lower extremities, the reflexes were bilaterally exaggerated but equal. A sustained ankle clonus was present on both sides. The left side of the body, including the face, showed a reduction to all forms of sensation, and the left hand showed a loss of stereognostic sense. White blood count 20,300; spinal fluid pressure was 300 Mm. of water, and showed four cells per cubic millimeter.

Operation.—June 2, 1938: Dr. L. Weinberger performed bilateral frontal and parietal trephines. No hematoma was found. The ventricles were tapped and the left was found to be larger than the right.

On June 3, 1938, Dr. F. C. Grant reflected a right frontoparietal bone flap. The

brain was under marked tension and the dura was opened rapidly. A subcortical lesion was indicated by widening of the gyri in the parietal and temporal lobes. A cannula was introduced in this area and it entered an abscess (Fig. 3). Two cubic centimeters of pus were obtained. The cannula was withdrawn. The dura was closed except at the base of the defect over the temporal lobe beneath the temporal muscle. The dura was then re-opened over the abscess and a transcortical incision was made down to the wall. The abscess was enucleated without rupture. The resulting cavity in the brain was packed with gauze soaked in azochloramine and an incision made in the scalp for its removal at a future time. The bone flap and scalp were replaced and the scalp closed by interrupted silk sutures.

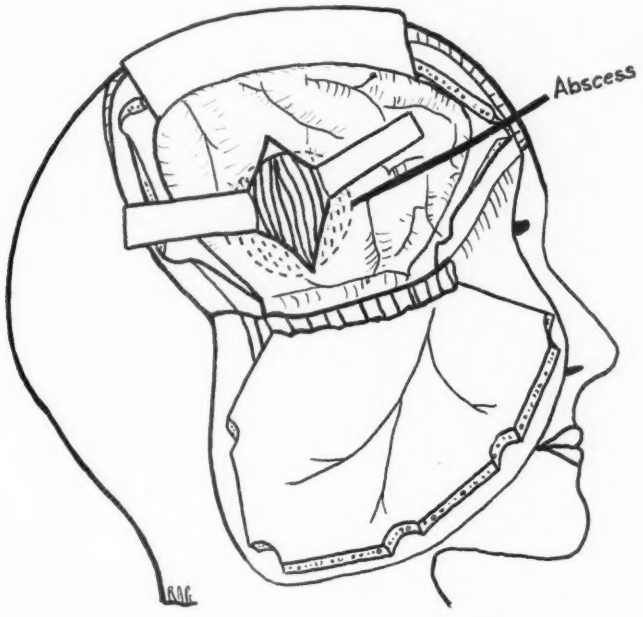

FIG. 3.—Case 3: Schematic diagram showing location of abscess in right parietotemporal lobes.

Pathologic Examination.—The abscess (Fig. 3) measured 5x4x2½ cm. The wall varied in thickness from 3 to 5 Mm. The pus was thick, greenish and foul-smelling. The organism was a gram-negative rod resembling *Haemophilus*. Microscopically, the wall consisted of a dense layer of collagen with few vessels and numerous polymorphonuclear cells.

The packing was removed on the second day but the wound continued to drain for 15 days and then healed. Pressure was controlled by daily lumbar punctures. The weakness on the left side of the body was more pronounced following operation but subsequently improved so that he was able to walk on the ninth postoperative day.

Subsequent Course.—Follow-up examinations up to the present time showed that the weakness of the left leg has cleared completely, the arm remains slightly weak and at about monthly intervals the patient has sensory jacksonian attacks in the left arm. Otherwise he feels well.

COMMENT.—The particular problem this case presented was to determine the course to be adopted when an abscess is tapped during a craniotomy. In this patient, the cannula was removed and the dura closed in order to maintain

pressure sufficient to keep the subarachnoid space closed. An opening was then made through the dura and brain down to the wall. The abscess was removed through this opening. To prevent possible spread of infection, the cavity was packed with gauze saturated with azochloramine. This course of treatment was effective in preventing meningitis in this patient.

Case 4.—*Synopsis: Symptoms of intracranial involvement for two weeks. Signs of intracranial hypertension. Ventriculogram. Right frontoparietal craniotomy and exposure of abscess. Subsequent removal of wall. Second admission: Removal of infected bone flap. Recovery.*

W. T., Hosp. No. 137004, male, age 11, was admitted to the Graduate Hospital, August 25, 1938, having been referred by Dr. Mark D. Grim, Oley, Pa. The patient had been in good health until eight weeks before admission to the hospital, when he developed

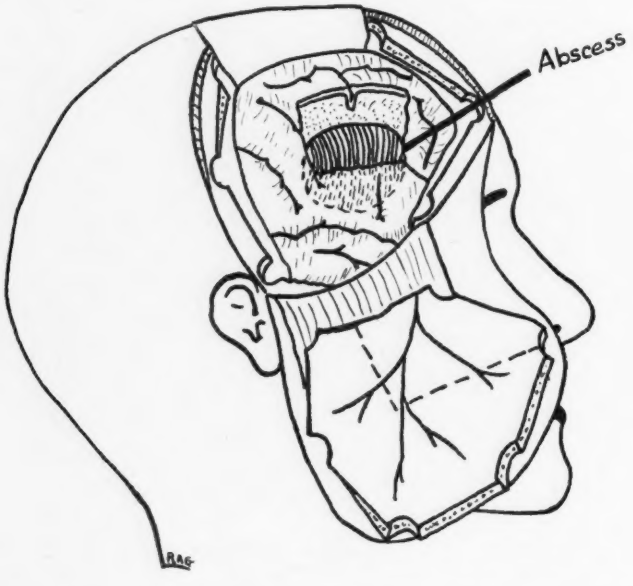

FIG. 4.—Case 4: Schematic diagram showing location of abscess deep in right frontal lobe.

frontal headache and a fever of 101° F. These symptoms continued for ten days, when he had a generalized convulsion. Following the convulsion all symptoms subsided. The patient was then well until two weeks before admission, when headache and vomiting began and continued up until admission to the hospital. One week before admission, examination of the eyegrounds showed papilledema of both optic disks.

Physical Examination.—This was essentially negative, and the neurologic examination was likewise negative except for a bilateral papilledema of four diopters. Visual fields were full. Barany examination was suggestive of an intracranial lesion. Roentgenograms of the skull and paranasal sinuses were entirely negative. White blood count 10,600; the blood Wassermann was negative.

Operation.—August 1, 1938: Dr. R. A. Groff performed a ventriculogram which demonstrated a lesion deep in the right frontal lobe. A right frontoparietal bone flap was reflected and no surface tumor was seen. A mass (Fig. 4) was felt 3.5 cm. below the surface of the frontal lobe in its posterior portion by an exploratory cannula. A block of cortex measuring 5 cm. square was removed over the lesion. A lumbar puncture needle was introduced into the lesion and 1 cc. of thick, yellow pus was obtained. Examination

of this pus showed many gram-positive Cocci. An attempt was made to enucleate the abscess but the wall was too thin. Sutures were then placed in the wall at its thickest portion. The dura and bone were removed over the lesion. Iodoform packing was placed in the cavity over the abscess and brought out through a separate overlying scalp wound. The remaining dura was sutured in position, the bone flap wired to the skull, and the scalp closed in two layers.

Postoperative Course.—The night of the operation, the abscess had pushed its way to the scalp edge, displacing the packing. A suction tip was inserted in a seepage point on the wall and two ounces of pus were removed. The opening in the wall was enlarged and packed tightly with iodoform gauze. Six days later, after signs of infection had subsided, the wall was teased out of its bed through the overlying scalp wound. During this maneuver the ventricle ruptured into the wound. This complication was treated by antiseptics to the surrounding scalp and application of sterile dressings. The cerebrospinal fluid leak continued for ten days and then stopped. Several days later pus was evacuated

FIG. 5.—Case 2: Postoperative photograph of patient showing well-healed scar following a left occipital craniotomy.

from above the right eyebrow and a sponge removed which had been left in at the time of operation. Both wounds healed subsequently and the boy was discharged 38 days after operation.

Subsequent Course.—On November 7, 1938, the patient was readmitted because of a draining sinus along the upper medial limb of the scalp incision. Roentgenograms of the skull demonstrated an osteomyelitis of the bone flap. The diseased bone was removed and the patient discharged 58 days later with wound healed except for a small area at the site of the previous drainage tract. Since discharge (Fig. 5) from the hospital following his second admission, the wound has healed and the boy remains well and is attending school.

COMMENT.—The surgical problem presented by this patient was similar in many respects to Case 3, except that the wall was too thin to permit enucleation at the time of the original operation. The fact that the abscess migrated to the surface indicated that the abscess would probably have delivered itself if sufficient time had been given. Kahn[3] has demonstrated this very dramati-

cally. The abscess cavity, however, was evacuated of its contents, packed with iodoform gauze and after evidences of active infection subsided, its wall was removed. The cerebrospinal fluid leak which followed was alarming but stopped under conservative treatment. The subsequent development of an infection in the bone flap was caused by an overlooked sponge.

Case 5.—*Synopsis: Severe head injury followed by symptoms and signs of focal brain disease with intracranial hypertension. Exploratory trephines and bilateral subtemporal decompression. Removal of three abscesses in stages through left subtemporal decompression.*

FIG. 6.—Case 5: Schematic diagram showing approximate location of abscesses.

J. R. C., Hosp. No. 35637, male, age 7, was admitted to the University Hospital, March 4, 1937, having been referred by Dr. B. L. Hull, Altoona, Pa. The patient had been well until nine weeks before admission when he fell from an embankment, striking the left frontal bone. He was unconscious at the time of the injury, subsequently regained consciousness and several hours later lapsed into unconsciousness. He remained in this state for 18 days. During this period a hematoma of the scalp developed over the left temple, which became infected, drained for six days and then healed. Five weeks before admission, or four weeks after injury, the patient had several generalized convulsions but improved and was out of bed. Up to one week before admission to the hospital, he had no complaints except that he tired easily. One week before hospitalization, or eight weeks after injury, the patient developed headache, nausea and vomiting. During the next few days these symptoms increased in severity. He became irritable, cried easily and developed weakness of the right face and upper extremity.

Physical Examination.—Temperature 99.3° F., pulse 102, respirations 26. The pa-

tient was drowsy but could be aroused easily. A scar was present over the left temple. Neurologic examination demonstrated weakness of the right lower face, bilateral choking of four diopters, a paralysis of the right upper extremity and a paresis of the left lower extremity. The reflexes on the right side were decreased and a Babinski was present on the right. White blood count 13,500; urine normal; spinal fluid pressure 750 Mm. of spinal fluid and the fluid contained six lymphocytes per cubic centimeter.

Operation.—March 5, 1937: Dr. F. C. Grant made trephines over the left frontal and parietal bones. No hematoma was found but the brain was under marked increased pressure. A cannula was inserted into the brain through the frontal bur opening and increased resistance was encountered at a depth of 3 cm. A third trephine opening was made in the upper limits of a subtemporal decompression. This opening was enlarged to the size of a decompression and the dura opened. During exploration, the patient strained and an abscess (Fig. 6) ruptured into the wound. About an ounce of pus was recovered. The wall was drained by rubber tissue and the wound left open. The pus contained Type I pneumococci on culture.

Postoperative Course.—Antipneumococcic serum was administered to the extent of 50,000 units. A cerebral fungus developed, which was amputated seven days after operation. That portion removed contained part of the abscess wall. During the following 24 hours the fungus increased greatly, so that a right subtemporal decompression was performed and the right ventricle tapped. The fungus was explored and the remainder of the original abscess wall removed together with two additional well walled-off abscesses.

The fungus increased in size and drained pus for a number of weeks. Intracranial pressure was reduced by lumbar punctures. These drainages were done as often as every 12 hours during the acute stages. Nine weeks after the last operation, the fungus was clean and had receded sufficiently to allow skin grafting over it. Two weeks later, or three months after admission, the patient was discharged from the hospital.

Pathologic Examination.—The abscess wall was composed almost entirely of collagen fibers arranged in parallel rows. Among these fibers were a few glial fibrils and numerous polynuclear leukocytes.

Subsequent Course.—One and one-half years later, the patient showed a useful right leg, a less useful right arm and a slight speech defect. These signs were much improved over those recorded when the patient was discharged from the hospital.

COMMENT.—The approach to the problem presented by this patient was different from that described in the preceding cases. In exploring for a subdural hematoma, an abscess ruptured into the field of a subtemporal decompression. The only alternative was the institution of drainage. A cerebral fungus quickly developed, was explored, and two well walled-off abscesses found and removed. The cause for the herniation was not only a focal cerebritis but two additional abscesses. Had this herniation not been explored, it is questionable whether this patient would have survived. Therefore, other abscesses as well as a cerebritis may be the cause of cerebral herniation.

Case 6.—*Synopsis: Symptoms and signs of intracranial hypertension for one month. Lesion localized to left side by shift of calcified pineal gland, as seen roentgenologically. Abscess in left frontal lobe tapped and drained. Subsequently refilled; attempted removal of wall by craniotomy; rupture of wall; later, removal of two-thirds of wall. Second abscess formed, drained, and complete removal of wall. Third abscess collection developed; wall opened and packed. Recovery.*

M. B., Hosp. No. 132888, male, age 17, was admitted to the Graduate Hospital, January 10, 1938, having been referred by Dr. Henry Dintenfass, Philadelphia, Pa. In August, 1937, this patient had had a right ethmoidectomy. Since that time he complained of generalized weakness. One month before admission to the hospital he developed severe frontal headaches and diplopia on looking to the left. The headaches became progressively

more severe and at the time of his admission, were almost continuous. One week before admission he had an attack of nausea and vomiting, associated with vertigo. From this time on he became increasingly more drowsy.

Physical Examination.—This was essentially negative. Temperature 99.6° F., pulse 60, respirations 24. Neurologic examination disclosed a patient mentally quite drowsy; showed a slight right lower facial weakness; a paralysis of the right external rectus; an increase in the reflexes on the left side; and the eyegrounds showed a papilledema of four diopters in both eyes. Roentgenologic examination of the skull demonstrated a calcified pineal gland, which was shifted from the left to the right side of the skull. The examination of the sinuses showed a densely clouded left antrum. *Preoperative Diagnosis:* Left frontal lobe brain abscess.

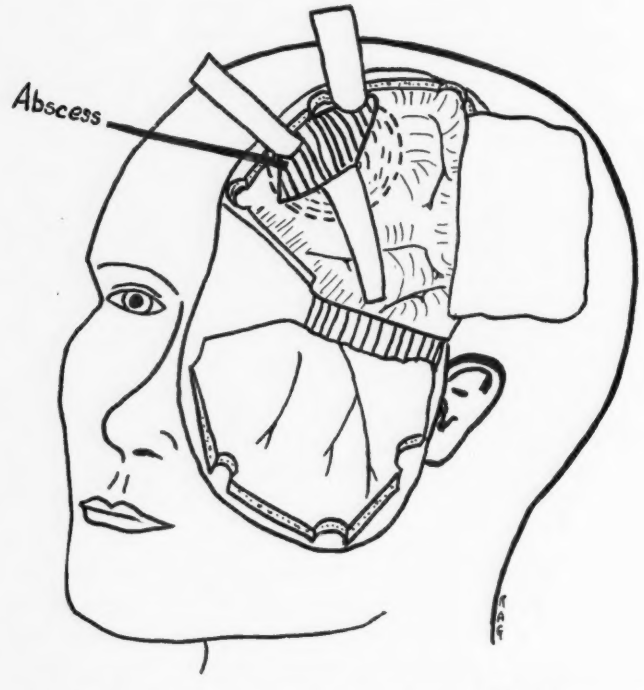

FIG. 7.—Case 6: Schematic diagram showing location of first abscess. The four other abscesses present in this patient were lateral and anterior to this abscess.

Operation.—January 12, 1938: Dr. R. A. Groff placed a trephine over the left frontal lobe and an abscess was palpated (Fig. 7) 2 cm. beneath the cortex. The wound was packed with iodoform gauze, and 24 hours later, the abscess was opened and a drainage tube inserted.

Postoperative Course.—The abscess continued to drain, and on the eleventh day following the institution of drainage, the eyegrounds showed a sudden increase in papilledema with fresh hemorrhages. A right subtemporal decompression was performed and the left frontal lobe explored through a clean trephine opening but no further pus was obtained. Eight days later, the tube was changed in the abscess and approximately 25 cc. of pus were obtained. Subsequently drainage ceased, the tube was removed, and the patient discharged 45 days after the initial drainage of the abscess.

Readmission.—April 15, 1938: Following discharge from the hospital, the patient remained in good condition up until the day before his present admission to the hospital, at which time he developed a continuous headache, became nauseated, and vomited several

times. Neurologic examination showed a right lower facial weakness and a definite increase in tension of the right subtemporal decompression.

Operation.—April 28, 1938: Dr. R. A. Groff performed a left frontoparietal craniotomy, under avertin anesthesia. The dura was opened and the abscess outlined by means of an exploratory cannula. An incision through the cortex, down to the wall, was made and an attempt undertaken to enucleate the abscess. The wall, however, was very firmly adherent to the falx. In attempting to break up these adhesions, the abscess ruptured. The hole in the abscess was then plugged, the bone flap removed in order to prevent it from becoming infected, and the scalp closed with silkworm sutures. An incision was made through the scalp in order to permit access to the abscess. This open wound was packed with iodoform gauze.

Subsequent Course.—Following this operation, no evidence of meningitis appeared. On the following day, the packing was removed and the abscess wall opened. Approximately 70 cc. of pus were removed. The abscess wall was then packed with iodoform gauze. Following this, drainage continued for several weeks, subsided, and the patient was discharged from the hospital 70 days following the institution of drainage.

Readmission.—September 5, 1938: The patient had developed symptoms and signs of a refilling of the abscess.

Operation.—September 6, 1938: Dr. R. A. Groff exposed the abscess by opening the scar which had been made previously. Sutures were placed in the wall. The wall was opened and two ounces of pus obtained. The wall was then packed with iodoform gauze. Four days after opening the abscess, the wall was dissected out. Unfortunately, only two-thirds of the wall was obtained. The remaining portion was firmly adherent to the falx. In removing the wall, the ventricle ruptured into the wound. This was treated by light packing. Twenty-six days later the wound was completely healed and the patient was discharged.

Readmission.—November 26, 1938: The day before admission the patient had a generalized convulsion. At the time of his admission, the examination was essentially negative except for a small drainage tract at the site where the abscess had been drained. Roentgenograms of the skull demonstrated what was thought to be a beginning osteomyelitis of the bone edge in this vicinity.

Operation.—December 3, 1938: Dr. R. A. Groff investigated the sinus tract, and the presumably infected bone was rongeured away. No evidence of osteomyelitis could be seen. The wound was closed with drainage. The drain was removed on the third day and the discharge ceased on the twelfth day. The patient was discharged 14 days following operation.

Readmission.—February 6, 1939: Two weeks before this admission, the patient contracted a cold and was sent to bed by the family physician who diagnosed the illness as "grippe." During succeeding days, decompressed areas became tense and began to bulge. After a period of ten days in bed, patient was pronounced cured of "grippe" and came to hospital because the decompression areas were "hard and tender."

Operation.—February 11, 1939: Dr. R. A. Groff performed a ventriculogram which demonstrated a lesion in the left frontal lobe. The scar, through which two previous abscesses had been drained and enucleated, was opened and the abscess wall exposed. The abscess was opened widely, evacuated and packed with iodoform gauze.

The cavity of the abscess stopped discharging two weeks later and began filling-in with fresh granulation tissue.

Three weeks after admission, the ventriculogram was repeated and the defect previously seen was still present but less marked. The patient was discharged six weeks after admission; wound not completely healed.

Readmission.—April 4, 1939: Four days before admission, the patient developed a "head cold" and subsequent to this, the decompressed areas became extremely tense. Headaches, nausea and vomiting brought him back to the hospital.

Operation.—April 20, 1939: The previous scar was opened by Dr. R. A. Groff. Two

abscess cavities were evacuated of one and one-half ounces of pus. These cavities were opened widely and packed with gauze.

Convalescence was interrupted by a generalized convulsion, nine days after operation. Three days later, packing was removed from cavities, since they were granulating-in rapidly. The decompressed areas became concave for the first time since treatment was started. The wound was practically healed when patient was discharged, May 6, 1939.

Since discharge from the hospital, the patient has remained symptom-free; the wound is well healed and decompressed areas are soft and sunken.

COMMENT.—The neurologic findings in this patient were indefinite but seemed to suggest a right frontal lobe lesion. Roentgenograms of the skull disclosed a calcified pineal gland displaced to the right. This evidence definitely placed the lesion on the left side and showed the value of roentgenograms of the skull and the presence of a calcified pineal gland.

The surgical treatment in this patient embodied three methods: Tap and drainage; enucleation; and marsupialization. Rubber drainage proved ineffectual. Enucleation was unsuccessful in the first abscess because the wall was firmly adherent to the falx. Only two-thirds of the wall of this abscess was removed. A second abscess was enucleated in stages. Marsupialization was employed twice for two additional abscesses. This method, described by Horrax,[4] has for its principle uncapping the abscess, fixing the wall to the scalp, and packing.

Case 7.*—*Synopsis: History of two head injuries followed by signs of increased intracranial pressure. Two tappings of right frontal lobe abscess. Craniotomy and removal of four encapsulated abscesses. Meningitis. Death.*

W. P., Hosp. No. 36666, male, age 20, was admitted to the Philadelphia General Hospital, April 26, 1937, having been referred by Dr. J. J. Curtin, Philadelphia, Pa. At age 15, the patient had a head injury with loss of consciousness, requiring hospitalization for 30 days. Several weeks following this injury, he became blind in the right eye; was hospitalized and subjected to an encephalogram. The vision in the right eye returned and the patient remained well until November 20, 1936. On this day, the patient struck his head against an iron pipe, became very dizzy, but did not lose consciousness. Two weeks later he developed headache. On December 22, 1936, he had an attack of unconsciousness during which he bit his tongue. This attack lasted 15 minutes and was not associated with convulsive movements. On January 4, 1937, a similar attack occurred and the patient was studied in another hospital for four weeks. He was discharged feeling well. On April 24, 1937, his headaches returned, and he was unable to hold anything on his stomach. His vision became "blurry." He was admitted to this hospital two days later with an increase in symptoms.

Physical Examination.—Temperature 99° F., pulse 50. Neurologic examination demonstrated definite clouding of consciousness; tenderness over the right temporal area; and slight blurring of the optic disks. The spinal fluid pressure was 21 Mm.Hg., and the fluid contained 281 cells; 24 per cent of polymorphonuclears and 76 per cent lymphocytes. Roentgenographic studies of the skull and sinuses were essentially negative except for "sclerosis" of the right sphenoid ridge.

Operation.—April 30, 1937: Dr. R. A. Groff made a trephine opening over the right frontal lobe just above the fascial attachment of the temporal muscle. An exploratory cannula encountered increased resistance at a depth of 3 cm., anterior and medial to the trephine opening. This mass was penetrated, and 30 cc. of greenish-yellow pus were

* Previously reported as Case 4, Brain Abscess of Undetermined Etiology, J. C. Yaskin, F. C. Grant and R. A. Groff, ANNALS OF SURGERY, 107, 492, April, 1938.

evacuated. The cavity was washed out with normal saline and the cannula left *in situ*. Forty-eight hours later, the cannula was removed because no drainage had occurred and normal saline washings of the cavity did not recover pus. The pus contained Type IV pneumococcus.

Postoperative Course.—The patient recovered rapidly and was discharged. Three weeks later he returned to the hospital with signs of refilling of the abscess. In attempting

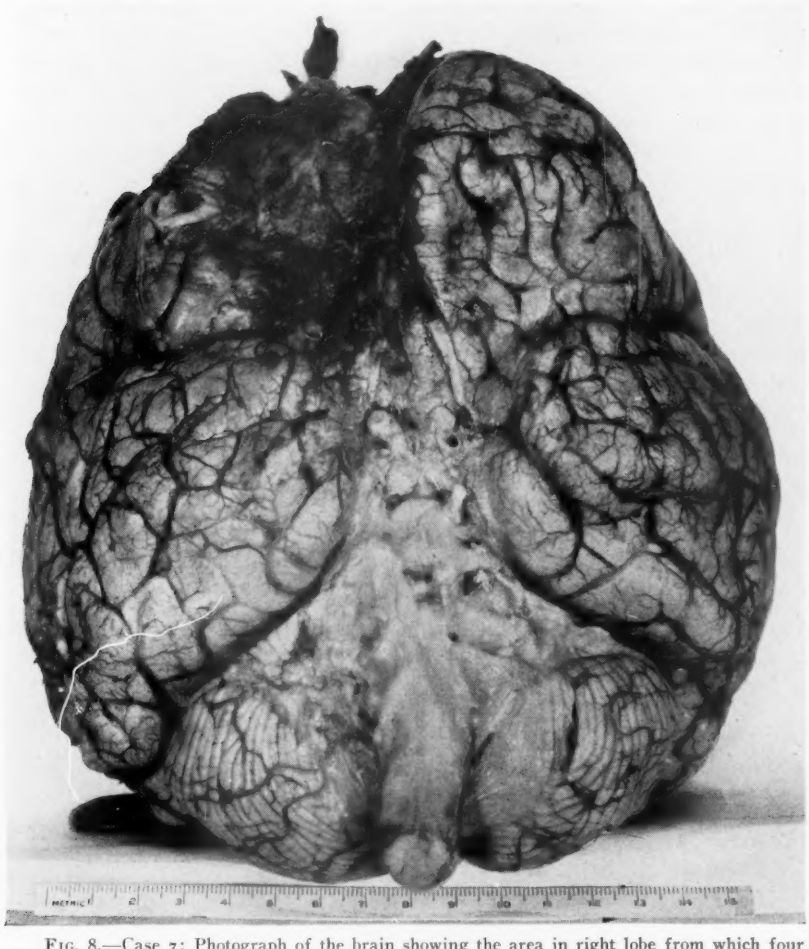

FIG. 8.—Case 7: Photograph of the brain showing the area in right lobe from which four abscesses were removed. Note the absence of infection over this area. The infection which terminated patient's life is shown confined to the base of the brain.

to evacuate the abscess for the second time, the cannula would not penetrate the abscess wall because of its thickness. A lumbar puncture was performed. Following this relief of pressure, the symptoms and signs subsided. Operation for removal of the wall was contemplated but the patient refused and left the hospital.

On July 5, 1937, he was admitted to the University Hospital, with symptoms and signs of refilling of the abscess in the right frontal lobe.

Operation.—July 6, 1937: Dr. F. C. Grant exposed the right frontal lobe. The abscess was identified, and just as its enucleation had virtually been completed, the wall ruptured. A second abscess was palpated in the temporofrontal lobes and, similarly, as

it was being enucleated, its wall ruptured. Two other abscesses were removed. During the operation a fracture in the posterior wall of the frontal sinus was exposed. The wound was cleansed, the dura closed and the flap and scalp replaced with drainage.

Postoperative Course.—Three days later, clinical evidence of meningitis developed, which was confirmed by purulent drainage from the wound and infected spinal fluid. The patient died 16 days after operation.

Autopsy.—The brain (Fig. 8) showed evidence of a marked basilar meningitis. The right frontal lobe, from which four abscesses had been removed, showed marked necrosis and destruction, with little evidence of infection. On section of the brain, there was a marked ventriculitis. Microscopic studies of the brain showed a diffuse purulent meningoencephalitis.

In addition, a fracture was demonstrated extending from the frontal to the ethmoid sinuses. The adjacent bone was infected and these sinuses contained pus. The conclusion from these findings was that the ethmoid and frontal sinuses were the primary focus of infection.

COMMENT.—The method of treatment employed in this patient seems to us to be ideal: First, initial tapping of the abscess in order to localize and estimate the thickness of the wall. Second, direct exposure and enucleation of the abscess. The unfortunate circumstance in this patient was the presence of more than one abscess, as well as the osteomyelitic area about the frontal and ethmoidal sinuses which would have, in all probability, continued to reinfect the brain.

ANALYSIS.—*Source of Infection:* The source of the infection from which the abscess in the brain developed was unknown in three patients, although in one of these, infected tonsils may have been the contributing factor. The remaining four patients developed a brain abscess during a chronic infection of the frontal and/or ethmoid sinuses. In these latter patients, roentgenographic studies demonstrated the sinus disease, but in no instance was there evidence of osteomyelitis of the bone in or about the sinuses.

Description of Abscess.—The locations of the abscesses were: Three in the frontal lobe; two in the temporal lobe; and one each in the occipital and parietal lobes. The depth of the abscesses in relationship to the surface of the brain varied. One lesion presented itself on the surface of the brain and, upon first inspection, was thought to be a meningioma. The remaining lesions were located beneath the surface, varying from a depth of 1 to an extreme distance of 4 cm.

Four patients had single abscesses, whereas the remaining three had multiple abscesses. Where multiple abscesses were present, they were in close relationship to each other and confined to one lobe.

Only one abscess had a stalk. This stalk extended from an infected fracture line involving the frontal and ethmoid sinuses, through the dura to the region of the abscesses in the frontal lobe. Four abscesses were present in this patient.

All abscesses had walls which were sufficiently firm to permit enucleation.

Diagnosis and Treatment.—The preoperative diagnosis in these seven patients influenced the method of treatment. In four cases (Cases 1, 2, 3 and

4), the primary diagnosis was brain tumor and in two of these cases (Cases 1 and 2), brain abscess was not considered a possibility. All four patients were subjected to a ventriculogram, when localization could not be made clinically, and then to a craniotomy. When the diagnosis of brain abscess had not been considered, the lesion was enucleated. That the lesion was an abscess was not appreciated until it was sectioned in the laboratory. When the diagnosis of abscess had been made preoperatively, a needle was introduced into the lesion after exposure had been made. Enucleation was performed immediately after the tapping in the one patient, whereas, in the second patient, the abscess was removed several days following the craniotomy.

A fifth patient (Case 5) was diagnosed a chronic subdural hematoma. After exploratory trephines had been made, a subtemporal decompression was carried out. An abscess ruptured into the operative field during this procedure. Subsequently, the wall of the ruptured abscess, together with two additional intact abscesses, was removed.

The diagnosis of the remaining two patients (Cases 6 and 7) was made preoperatively. After preliminary tapping and rubber tube drainage had been performed unsuccessfully, both were subjected to a craniotomy. The wall of the abscess was so firmly adherent to the falx in one patient (Case 6) that it ruptured while attempting to remove it. Part of the wall was removed a few days later. Several months afterward, a second abscess became apparent; this was drained and the wall removed completely. Approximately six months later, two abscesses were localized and treated by marsupialization.

The last patient (Case 7) had four abscesses, two of which ruptured during removal. One of these abscesses had a stalk which connected with an infected fracture line in the frontal and ethmoid sinuses. This stalk was disturbed during the operation and meningitis terminated the patient's life. This is the only death in this series.

Complications.—The only complication in the four patients with a single abscess was an osteomyelitis of the bone flap. This unfortunate circumstance could have been avoided, since it was the direct result of the operator's (R. A. G.) overlooking a sponge. The complication which arose in the treatment of the patients with multiple abscesses was meningitis in one patient. This was the result of a disturbance of the stalk of one of the abscesses and, in addition, the rupture of two abscesses during removal. In another patient, a cerebral herniation resulted, and within this mass two walled-off abscesses were found and removed.

COMMENT.—The smooth, rapid and uncomplicated convalescence in the four patients with single abscesses, if one overlooks the avoidable complication of osteomyelitis of the bone flap in the one patient, commends this procedure as the most satisfactory treatment for brain abscesses which have a firm wall. The complications of cerebral herniation, cerebral fungus, and an alarming increase in intracranial pressure are avoided. The constant wound care necessitated, when drainage methods are employed, is eliminated. The postoperative care is reduced to the management of a patient who has been operated

upon for a brain tumor, and the morbidity rate in these patients, at the present writing, is extremely low. These facts justify the surgical procedure of enucleation of a well walled-off brain abscess.

The term "capsule" has been purposely discarded, because the meaning conveyed by it is a covering for organs, or certain tumors. The layer of tissue about an abscess is a barrier to enclose or confine the infection. This barrier or wall is not a capsule nor is it referred to as such in abscesses in other parts of the body. We are in accord with Atkinson,[5] that the tissue surrounding an abscess should be called the "wall."

The enucleation of a brain abscess by direct exposure depends upon the firmness of the abscess wall. Therefore, this fact must be known before enucleation is contemplated. The most practical way in which this information can be determined is by exploration with a cannula through a trephine opening. If the cannula imparts the information that the wall is firm, enucleation of the abscess by direct exposure can be carried out immediately. On the other hand, if the cannula passes through the wall without much resistance, it will be necessary to adopt measures to allow more time, in order that the wall of the abscess may become firmer and thicker. These measures may be one of several; repeated tappings of the abscess, such as was done in Case 7; or repeated tappings and a subtemporal decompression, in order to control increase in intracranial pressure and save vision, as was carried out in Case 6. A craniotomy may be performed without opening the dura, as suggested by Vincent. The latter method was utilized in a modified form in Case 4. In this patient, after the abscess had been exposed, it was found that the wall was not thick enough to permit enucleation. The abscess was removed several days later.

Tapping the abscess gives the additional information of the type of organism which is responsible for the infection. If this organism is one of those susceptible to the several chemotherapeutic agents, they can and should be given, as suggested by Rowe[6] and Bucy.[7]

Multiple abscesses of the brain present a different problem. The three cases presented here were more or less ideal in that the abscesses were grouped together. Two of these patients were treated successfully; the third died of meningitis. The difficulty with this problem is that the walls of the several abscesses are not in the same stage of development. Thus, if a preliminary tap is made and the cannula strikes an abscess with a firm wall, and direct exposure discloses several lesions, one or more of them may not have a wall which is firm and sufficiently thick to remove without rupture.

Abscesses resulting from penetrating wounds and direct extension of the infection from either an infected mastoid or frontal sinus cannot be treated by this method. The one patient in this series (Case 7), whose lesion had a stalk leading from an infected frontal and ethmoid sinus, died from meningitis as a result of disturbance of this tract. The methods of King,[8] Horrax[4] and Kahn[3] are more applicable for treating this type of pathology.

SUMMARY

The records of seven patients with brain abscess, in whom the surgical treatment was enucleation by direct exposure, have been presented.

The reasons for considering the method of enucleation of firmly walled-off brain abscesses as the most satisfactory form of treatment have been given.

The problems which arose in connection with this method of treatment have been discussed, and an outline has been given for the management of patients in whom the diagnosis of brain abscess has been made.

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THE TREATMENT OF ADDISON'S DISEASE BY THE IMPLANTATION OF SYNTHETIC HORMONE*

WARFIELD M. FIROR, M.D.
BALTIMORE, MD.

IT IS highly probable that human beings have died from destruction of their adrenal glands since the beginning of the race, but the clinical picture incident to this destruction has been recognized for only 84 years, and the physiologic alterations that are induced by adrenal disease have been understood for less than 10 years. Finally, a crystalline substance which rectifies these alterations, which abolishes the symptoms, and prevents death, has been available for less than two years. It is the purpose of this communication to tell of 17 patients who have been given this specific substance in the form of pellets implanted subcutaneously.

Since 1855, it has been known that destructive lesions of the adrenal glands cause weakness, loss of weight, hypotension, digestive disturbances, pigmentation and death. An excerpt from Addison's[1] original paper will suffice to show how clearly he grasped the essential features of the syndrome resulting from adrenal insufficiency:

"The patient, in most of the cases I have seen, has been observed gradually to fall off in general health; he becomes languid and weak, indisposed to either bodily or mental exertion; the appetite is impaired or entirely lost; the whites of the eyes become pearly; the pulse small and feeble, or perhaps somewhat large, but excessively soft and compressible; the body wastes without, however, presenting the dry and shriveled skin and extreme emaciation usually attendant on protracted malignant disease; slight pain or uneasiness is from time to time referred to the region of the stomach, and there is occasional actual vomiting, which in one instance was both urgent and distressing; and it is by no means uncommon for the patient to manifest indications of disturbed cerebral circulation." . . . "We discover a most remarkable and, so far as I know, characteristic discoloration taking place in the skin—sufficiently marked, indeed, as, generally, to have attracted the attention of the patient himself, or of the patient's friends.

"The disease develops in the third or fourth decade of life, usually quite insidiously, with adynamia and apathy. To these are added disturbances of the digestive tract (constipation, often alternating with diarrhea), and pigmenting of the skin and mucous membranes: the patients succumb under a gradually increasing cachexia, not rarely with stormy terminal manifestations; autopsy almost always shows disease of both suprarenals, mostly tuberculous caseation."

Addison not only gave to the world a concise description of the disease

* Read at the Fifty-second Annual Meeting of the Southern Surgical Association, Augusta, Ga., December 5, 6, 7, 1939.

which bears his name, but he stated, quite correctly, that any chronic lesion destroying the adrenals would give rise to this syndrome.

In 1875, Greenhow[2] drew attention to an additional characteristic of the symptomatology of Addison's disease. His outstanding paragraph states: "The asthenia, the constitutional symptoms generally, and the change of colour in the skin are all, it is true, progressive, but not steadily so. The course of the disease, on the whole, is slow and chronic; but it is subject to alternate exacerbations and remissions, usually in some degree dependent upon favourable and unfavourable circumstances but sometimes also, apparently, quite independent of them. During the remissions, strength is in a great degree recovered, appetite improved, sickness abated; the discolouration becomes paler, and, above all, the patient's whole aspect bespeaks that a heavy weight has been lifted from his head. After each fresh exacerbation, however, the patient remains upon a somewhat lower level than during the previous remission. The recovery of strength and the abatement of other symptoms is less marked, and the skin, though paler than during the last exacerbation, is yet visibly darker than before it. Similar alternations may occur several times before the onset of the fatal paroxysm, but on each occasion the patient takes at least one downward step that he never regains."

There were no other noteworthy milestones in the understanding of this disease until 1894, when Oliver and Schaefer[3] obtained a strong pressor substance from extracts of the adrenal medulla. This discovery initiated a period of intensive experimental activity, culminating in the isolation of epinephrine in 1904. It was soon realized, however, that the symptoms of Addison's disease were not ameliorated by the administration of epinephrine. Later, it was discovered that the removal of one adrenal, accompanied by the destruction of the medulla of the remaining gland, did not evoke evidence of adrenal insufficiency. From this experiment it was clear that the adrenal cortex is the part of the gland essential for life; thereafter, sporadic efforts were made to extract from the adrenal cortex a substance or substances which would sustain life in adrenalectomized animals. In 1929, a tremendous impetus was given to this search by the announcement of Pfiffner and Swingle[4] that they had succeeded in preparing a potent cortical extract. Immediately, work was begun in several large medical centers, and scientific publications soon reflected a revival of interest in the adrenal cortex. During the next two years three sets of workers claimed that they had extracts capable of counteracting the effects of adrenal insufficiency in animals. These various cortical extracts, however, gave most disappointing results when given to patients suffering from Addison's disease. The preparation of these extracts was expensive, and the standardization was time-consuming, since each lot had to be separately assayed; but, above all, the effects on the patients were variable and unpredictable. Snell,[5] at the Mayo Clinic, in 1933, concluded that the life expectancy of patients treated with the several cortical extracts then in use had been only slightly prolonged. Occasionally, patients seemed to show surprising and sustained improvement; but one questions the ad-

visability of attributing these results to the use of an extract, for had these extracts contained a potent cortical substance, then all patients with adrenal insufficiency should have benefited from them. To one who was actively participating in this field of investigation, it seemed that the clinicians suddenly began to recognize many more instances of the disease than ever before. Undoubtedly, some of the therapeutic results accredited to the various cortical extracts, between 1931 and 1933, were obtained from patients suffering from asthenia of nonorganic origin.

The renewed interest in the experimental and clinical evidences of adrenal insufficiency produced two discoveries of fundamental importance. The first of these came in 1933, when Loeb[6] pointed out the beneficial effect of sodium chloride in the treatment of adrenal insufficiency. The studies of Loeb and his co-workers made it clear that an animal with serious impairment of cortical function loses an abnormal amount of sodium and chloride ions in the urine. Such an animal has a concomitant increased urinary output. These alterations are reflected in the condition of the animal's blood, which shows low values for sodium and chloride and coincident hemoconcentration. Loeb showed that the ingestion of relatively large amounts of sodium salts was beneficial to patients with Addison's disease. Subsequently, Truszkowski and Zwemer[7] demonstrated the advantages of a diet having a low potassium component.

The second fundamental discovery in this field of research came four years later, when Steiger and Reichstein[8] announced the synthesis of a steroid compound capable of preventing death from adrenal insufficiency. This crystalline compound was desoxy-corticosterone acetate. One year later, Reichstein and von Euw[9] isolated this same substance from beef adrenal glands, thus proving its natural occurrence. This is the first instance in history in which a hormone has been synthesized before it had been isolated from its natural source.

Dr. George W. Thorn,[10] of the Department of Medicine of the Johns Hopkins Hospital, was fortunate enough to be given some of Reichstein's crystalline substance. At first this was tested on dogs, which the author had bilaterally adrenalectomized under spinal anesthesia. It was found that a single daily injection of 1 to 1½ mg. of desoxy-corticosterone acetate maintained a 10 Kg. adrenalectomized dog in good health, even though the animal was being kept on a diet low in sodium chloride and relatively high in potassium. When the injections were stopped, the animal showed a prompt diuresis associated with increased excretion of sodium and chloride and a decreased excretion of potassium, moderate hemoconcentration, and, in addition, it developed weakness, digestive disturbances, and loss of weight. When the administration of the crystalline substance was resumed, these changes were reversed and the animal soon returned to a normal condition. Subsequently, pellets composed of crystals of desoxy-corticosterone acetate were placed subcutaneously in bilaterally adrenalectomized dogs. It was found that this form of therapy maintained the animals in excellent health, if the daily hormone requirement of each dog had been previously determined and sufficient pellets

were implanted to meet these requirements. Removal of the pellets invariably brought on typical metabolic alterations and signs of adrenal insufficiency. By weighing the pellets before implantation and upon removal, the amount of hormone absorbed was ascertained. The rate of absorption was found to be from 0.25 to 0.40 mg. per day. This relatively slow absorption rate is due to the hardness of the pellets and to the slight water solubility of the compound. It did not vary in different animals. The comparison of the utilization of the cortical hormone when given by daily injections in oil, or when supplied by pellets, showed that the latter form of administration was about 25 per cent more efficient than the former.

The success of pellet therapy in experimental animals prepared the way for the use of the method in patients with Addison's disease. Since September, 1938, Dr. George Thorn and I have treated 17 patients by the implantation of pellets of desoxy-corticosterone acetate. All these patients have been studied on the Metabolic Ward of the Johns Hopkins Hospital. In every instance, the daily requirements of the hormone in sesame oil have been determined before the pellets were inserted. It has been found that 0.5 mg. of the hormone in oil, given by daily injections, requires a tablet weighing 125 mg. It is important to have the patient on standard conditions and to compute each patient's requirement before implanting the pellets. Our usual plan of procedure is to study the patient under basal conditions, and then, after this, to add 10 Gm. of sodium chloride to the patient's diet. We do not modify the potassium content of the diet. On this regimen we give a single daily injection of "Percorten" (Ciba) varying from 2 to 10 mg., depending upon the patient's needs.

As evidence of adequate treatment we require the patient to maintain: (a) Optimum body weight; (b) normal blood pressure with normal plasma volume; (c) positive sodium and chloride balance; and (d) normal concentration of plasma electrolytes. When the hormone requirements of a patient are accurately established, we discontinue the injections in oil and insert subcutaneously a sufficient number of pellets to supply a patient's needs.

The desoxy-corticosterone acetate is sterilized by dissolving it in hot acetone, and subsequently passing it through a Seitz filter. The crystals are treated in an autoclave for a few minutes at low pressure, and for additional protection they are placed in ether just prior to insertion. We are still working on the sterilization of this product and do not think that the most efficient means has as yet been determined.

All operations have been performed under procaine anesthesia. The pellets have been placed in the infrascapular region. Owing to the susceptibility of addisonian patients to infection, we have insisted on following the most rigid aseptic technic. The operations are performed in the general operating room. In three patients it has been necessary to insert tablets in two sites, and for these we have used a separate table of sterile equipment for the second operation. In one patient we inserted as many as 12 pellets through a single incision.

Operative Technic.—The incisions are carried well into the subcutaneous fat, and by inserting and spreading blunt scissors a number of pockets are made in this layer. These pockets radiate from the incision like spokes in a wheel, and are approximately 5 cm. long and 1 cm. wide. When hemostasis has been completed, a nasal dilator is introduced into each pocket and opened so as to facilitate the insertion of a pellet. By tying a black silk suture with an attached silver clip, a particular pellet may be marked for future identification and removal.

In the 17 patients thus treated from September, 1938 to October, 1939, we have made over 30 implantations. In no instance has there been any evidence of infection. All the wounds have healed *per primam*, and not a single pellet has been extruded from the wound.*

None of the patients has complained of pain or soreness after the introduction of the hormone. By removing and weighing some of the pellets we have established an absorption rate of this compound in human beings. It has been remarkably constant in all the patients and amounts to 0.3 mg. per day for each pellet weighing between 100 to 125 mg. This rate is somewhat dependent upon the softness of the pellet and upon the surface area. It is not dependent upon the physiologic needs of the patient, for we have found that patients with severe adrenal insufficiency do not absorb desoxy-corticosterone acetate faster than those who have only a slight insufficiency. Our experience has shown that the pellets placed in the 17 patients, referred to above, have met the patient's requirements for cortical hormone for periods ranging from four to nine months. Several of the patients have had their second implantation, and in one of these there is reason to believe that there has been partial regeneration of cortical tissue.

The results obtained in the 17 patients under observation may be summarized as follows: All but two of the group have returned to full activity, and are working as strenuously as they did before their illness began. One patient nursed her husband through a severe infection, in addition to carrying on with her household duties. Another patient was able to have a tuberculous kidney removed with the help of a few injections of the hormone in oil through the postoperative period. Every patient has gained weight, as is shown in Table I. The average gain for the group has been 5.4 Kg. The improvement in both diastolic and systolic blood pressure has been uniform (Table II).

In none of the 17 has there been any suggestion of hypertension, which we attributed to the careful computation of hormone requirements before the implantation was performed. All the patients had maintained a positive sodium and chloride balance. They have kept normal concentrations of potassium, sodium and chloride ions in the blood plasma. Similarly, the hematocrit and plasma volume determinations returned to normal after treatment was begun and have remained so. Some of the patients have shown a decrease in the pigmentation of their skin, but none of them has lost all the pigmentation. As

* One clinician, who has implanted pellets of testosterone by the punch technic, has reported that a fair number of the pellets are subsequently eliminated through the wound.

far as we can tell, no patient in the group has shown any untoward effect from this form of treatment.*

TABLE I

THE EFFECT OF SYNTHETIC ADRENAL CORTICAL HORMONE ON BODY WEIGHT

Patient	Before Treatment	Present Weight	Duration of Treatment
M. M.	51.3 Kg.	54.0 Kg.	380 days
P. W.	51.0 Kg.	55.4 Kg.	379 days
E. V.	54.4 Kg.	64.0 Kg.	377 days
C. N.	55.9 Kg.	64.3 Kg.	372 days
F. M.	61.2 Kg.	65.4 Kg.	370 days
J. Z.	39.3 Kg.	44.5 Kg.	363 days
A. S.	57.4 Kg.	59.2 Kg.	328 days
J. S.	55.2 Kg.	70.1 Kg.	290 days
J. H.	63.2 Kg.	69.7 Kg.	271 days
D. B.	64.7 Kg.	72.6 Kg.	261 days
I. B.	48.0 Kg.	52.1 Kg.	234 days
S. W.	58.5 Kg.	64.5 Kg.	207 days
R. W.	71.2 Kg.	73.3 Kg.	181 days
Y. E.	51.9 Kg.	52.1 Kg.	157 days
W. B.	41.2 Kg.	41.6 Kg.	150 days
R. M.	49.6 Kg.	54.3 Kg.	123 days
F. H.	59.5 Kg.	68.6 Kg.	65 days

TABLE II

THE EFFECT OF SYNTHETIC ADRENAL CORTICAL HORMONE ON BLOOD PRESSURE

Patient	Before Treatment	Present Blood Pressure	Duration of Treatment
M. M.	83/72	118/94	380 days
P. W.	96/74	130/80	379 days
E. V.	94/66	142/98	377 days
C. N.	94/60	134/76	372 days
F. M.	90/56	134/98	370 days
J. Z.	94/52	136/92	363 days
A. S.	110/70	145/95	328 days
J. S.	92/70	140/90	290 days
J. H.	98/60	130/90	271 days
D. B.	108/68	135/85	261 days
I. B.	110/74	150/98	234 days
S. W.	120/76	124/78	207 days
R. W.	108/70	126/76	181 days
Y. E.	102/74	140/90	157 days
W. B.	90/55	122/76	150 days
R. M.	98/60	136/112	123 days
F. H.	95/54	130/90	65 days

* Detailed protocols of the first six patients have been reported in the Bulletin of the Johns Hopkins Hospital,[11] and a comprehensive report of the entire group is in the process of publication.

Before leaving the subject it is important to sound a word of warning concerning the use of desoxy-corticosterone acetate. Since the rate of absorption depends upon the consistency and surface area of the pellets, one can easily imagine that improperly prepared pellets might crumble. This accident would result in a rapid absorption of a large amount of this potent hormone. Furthermore, it is wise to remember that many of the patients suffering from Addison's disease are really very sick, and the mere introduction of pellets is not sufficient in their care. They should be under careful supervision for at least two weeks after the implantation of the pellets. At present, the Government has not authorized the sale of desoxy-corticosterone acetate, nor has it been accepted by the Council on Pharmacy and Chemistry of the American Medical Association.

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Discussion.—Dr. Harvey Stone (Baltimore, Md.): I think everyone here must have been impressed, first, with the masterly presentation by Doctor Firor of this important subject and the clear historic review of the sequence of development up to the present time. I am sure also we are impressed with this great addition to the therapeutic possibilities of treatment of deficiency disease by this new method, and I think we can all gather a lesson from it that advance at the present time in the complicated problems surgery now presents requires the concerted efforts of the physiologist, chemist and surgeon working together. It is no longer a question of a man working by himself with any hope of success. Doctor Firor has played an important part in this sequence of development.

I should like to refer to some work Doctor Owings and myself have carried on along somewhat similar lines. A few years ago we presented before this Association some experimental work in the transplant of living endocrine organs. At that time we were entirely concerned with the thyroid and parathyroid. Since then we have continued with that work, and this may be a favorable opportunity to make a brief report of our very scanty efforts in transplant of the adrenal cortex. We have done three cases. Without going into detail, two of these cases showed notable and definite improvement. One lived for nine months after transplant and was able to return to work, following a rise of blood pressure comparable to those reported by Doctor Firor. He died of pulmonary tuberculosis. A second patient lived for two and one-half years after the first transplant, and was able to return to work with a com-

parable rise of blood pressure similar to those reported by Doctor Firor. At the end of about two years he showed some failure of improvement and came back for a second graft, was again improved and about half a year later acquired an acute influenzal pneumonia and died. On the third case we are not able to present much information. The patient left Baltimore and we lost track of him. We know he died a number of months later and we have nothing to say about the improvement, as to whether it was marked or not.

So far as the immediate effect of the graft is concerned, I think it is no better than pellet implantation of synthetic hormone which Doctor Firor discusses, and it may be that improvement in these cases was not so much due to the success of the graft as to the slow release of the hormone in the grafted tissue. It is difficult for me to assume that we grafted sufficient tissue to give improvement for two years, and the evidence suggests that the grafts survive for a certain period of time and then disappear.

One last word: This work still falls short of the ideal in supplying the patient with a living graft which will not need replacement as is obviously necessary with the pellet method of Doctor Firor, and I think everyone will agree the ideal remedy would be something that will last as long as the recipient survives.

Dr. George J. Heuer (New York, N. Y.): I also should like to congratulate Doctor Firor on his splendid piece of experimental and clinical research. I am not sufficiently familiar with the subject to discuss it, but I would like to know whether Doctor Firor has observed any ill effects from implanting the pellets in the tissues or whether he anticipates any danger from their use.

Dr. Warfield M. Firor (Baltimore, Md., in closing): I am glad Doctor Heuer asked that question. There is a real danger in this form of treatment, inasmuch as the rate of absorption depends upon the consistency of the pellet; if one uses a soft pellet that crumbles easily, a patient will absorb an enormous amount of this potent hormone very quickly. We have had such a case called to our attention. The patient's blood pressure went to 200 and there were signs of impending cardiac failure. The patient came to Baltimore and I had to take out most of the pellets because this patient, having received soft pellets, was absorbing them too rapidly. One has to be very cautious; and we do not allow any of these patients to go home until they have been in the hospital for about two or three weeks after the pellets have been implanted. In one woman the absorption rate was too rapid, and I took out two of the 12 pellets I had put in before I let her return home. We do not feel that this form of treatment is ready for general use, and we are not absolutely certain that we have determined the best way of sterilizing these pellets.

We dissolve the hormone in hot acetone and then pass it through a Seitz filter. The pellets are autoclaved at low pressure and finally washed in ether just before implantation. The Ciba Company, which has undertaken the preparation, has had a little difficulty in making pellets of uniform consistency. The Government will not permit desoxy-corticosterone acetate to be released, and the Committee on Pharmacy of the American Medical Association has not yet given its approval that this substance is a standard substitute for adrenal insufficiency.

CYSTS, SINUSES AND FISTULAE OF THE THYROGLOSSAL DUCT *

RESULTS IN TWO HUNDRED AND NINETY-THREE SURGICAL CASES

JOHN DEJ. PEMBERTON, M.D.,
DIVISION OF SURGERY,
AND
LEONARD K. STALKER, M.D.
ASSISTANT SURGEON, THE MAYO CLINIC,
ROCHESTER, MINN.

OF the congenital anomalies commonly encountered in the cervical region, perhaps none have resisted surgical correction for so long a time as have the cysts, sinuses and fistulae arising from a persistent thyroglossal duct. The reasons for this are not altogether clear. Since the nature of the developmental defect has long been well understood and since the problem of treatment offers little technical difficulty, it seems incredible, in retrospect, that surgeons continued to employ with so little success inadequate measures until 1920, when Sistrunk[4] advised the excision of the entire thyroglossal tract and devised for this purpose a safe and thoroughly satisfactory operation. Sistrunk was not one of the first to perform a radical procedure in these cases. Schlange,[3] in 1893, and Durham,[1] in 1894, cognizant of the fact that unsuccessful attempts at cure were due to the incompleteness of the operative removal of the tract, divided and removed a segment of the hyoid bone and dissected the duct to the root of the tongue. Later Eliot,[2] Spencer[6] and others advocated similar procedures. However, Sistrunk, recognizing that it is not always possible to trace the upward extension of the tract, even in cases in which the sinus has been injected with methylene blue, devised a procedure that would insure the complete removal of the entire tract and advocated its employment as routine.

A thyroglossal duct cyst is a retention cyst which arises in a patent portion of the vestigial thyroglossal tract and occurs anywhere in the midline along its pathway from the base of the tongue to the region between the hyoid bone and the thyroid gland. Clinically, the site of the cyst is far more common below the level of the hyoid bone than above it. Frequently the cyst ruptures through the skin spontaneously or is incised; an intermittent draining channel which may end blindly in the tissues, that is, a sinus, may result from rupture or the channel may run uninterruptedly to enter the mouth through the foramen caecum, that is, a fistula.

Knowledge of the origin and development of the thyroid gland simplifies the understanding of the surgical treatment of thyroglossal duct cysts. The

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principal, if not the only, primordium of the thyroid gland arises as an evagination or outpocketing in the ventral wall of the primitive pharynx between the first and second pharyngeal pouches. This position is marked in adult life by the foramen caecum at the base of the tongue behind the apex of the "V"-shaped row of the circumvallate papillae. The primitive thyroid structure descends in the midline through tissue which later becomes the hyoid bone. Inasmuch as the hyoid bone develops either simultaneously with, or later than, the descent of the thyroid gland, the thyroglossal duct may vary in its relation to the hyoid bone. The tract lined with epithelium produced by this descent normally disappears between the fifth and eighth weeks of fetal life, but occasionally fails to become obliterated. This failure of obliteration of either a portion of, or of the entire, thyroglossal duct subsequently may lead to the formation of a thyroglossal duct cyst, sinus and rarely a fistula (Fig. 1).

The majority of operations for the treatment of these conditions are unsuccessful unless the entire tract running from the cyst to the foramen caecum is removed. Since Sistrunk[4, 5] proposed this operative technic for radical removal of a thyroglossal duct cyst and its tract in 1920, his operative procedure has been employed in the surgical management of the majority of the cases at the Mayo Clinic. The operation as proposed by Sistrunk and employed by us, with a certain few minor modifications, is as follows:

Under general anesthesia, a transverse incision two or three inches (5 to 7.5 cm.) in length is made at about the level of the hyoid bone (Fig. 2, inset). When a sinus is present it may be injected with methylene blue to outline its course and an elliptic transverse incision made to include the sinus. The skin, subcutaneous tissue, and platysma muscle are reflected. The cyst or sinus tract will usually be found lying on the thyrohyoid membrane. It is dissected free from the surrounding tissues up to the hyoid bone. The relation of the tract to the hyoid bone is variable, usually passing through or beneath the bone, but occasionally passing above it. For this reason, to insure complete removal of the tract at this point and to facilitate exposure above this point, the central portion of the hyoid bone is freed above and below and the midportion, measuring about one-quarter inch (0.63 cm.) in length, is removed with bone forceps. As a rule the dissection up to this point is carried out without great difficulty, but above this the tract is usually so small and friable that it is broken off easily and is difficult to remove. For this reason no attempt is made to isolate the tract, but the duct and tissues surrounding it for a distance of 3 or 4 Mm. on all sides are cored out through the muscles of the tongue to the foramen caecum (Fig. 2). This can be done without difficulty as Sistrunk has shown that the pathway of the duct corresponds to a line drawn at an angle of 45 degrees backward and upward through the point of intersection of lines drawn horizontal and perpendicular to the center of the hyoid bone (Fig. 1). This dissection can be facilitated by placing the index finger of the left hand in the patient's mouth and pushing the foramen caecum upward and forward (Fig. 3). A portion of the hyoid bone, portions of the raphe of the mylohyoid muscles, and portions of the

geniohyoid and genioglossus muscles are removed. In cases in which the duct is patent above the hyoid bone the formen caecum is also removed (Fig. 4).

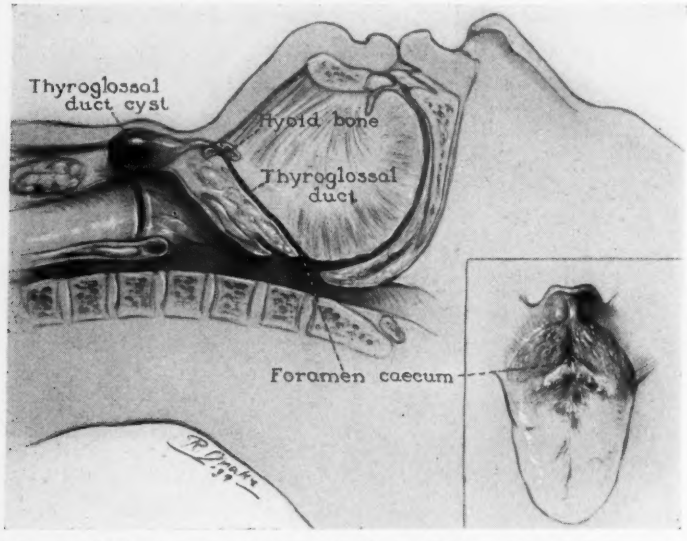

FIG. 1.—Sagittal section of head showing position of thyroglossal duct. Inset: Dorsum of tongue with foramen caecum.

but in others in which the cyst seems to be confined entirely to the subhyoid region it has been satisfactory to stop the dissection just at the foramen caecum

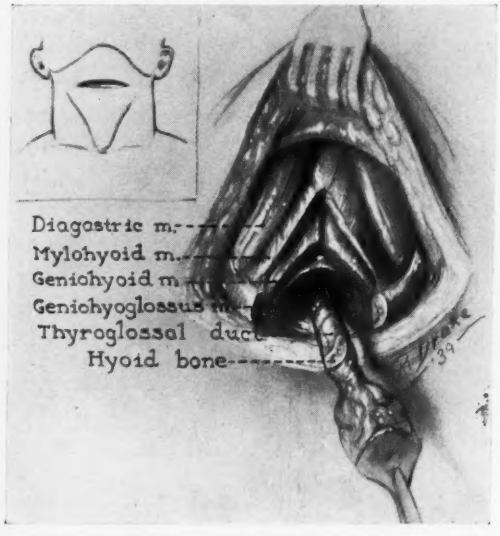

FIG. 2.—Dissection of thyroglossal duct tract. A segment of hyoid bone has been removed and duct with surrounding tissue is dissected through the muscles of the tongue. Inset: The incision.

without removing it and consequently without actually entering the mouth. There is, however, no serious objection to entering the mouth. The opening

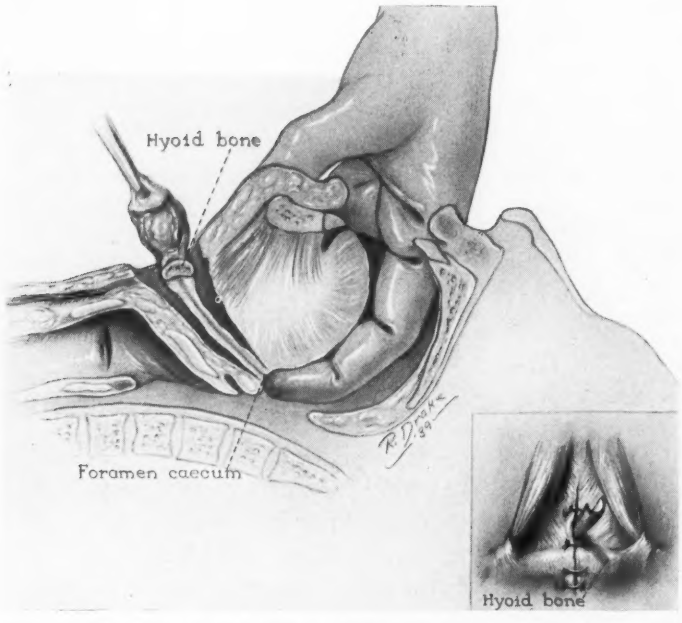

FIG. 3.—Finger in mouth locating foramen caecum and elevating tract. Inset: Closure of wound.

through the muscles of the tongue is closed with interrupted sutures of catgut placed superficially to avoid the hypoglossal nerves. The edges of the hyoid bone are brought together with stitches through the periosteum (Fig. 3,

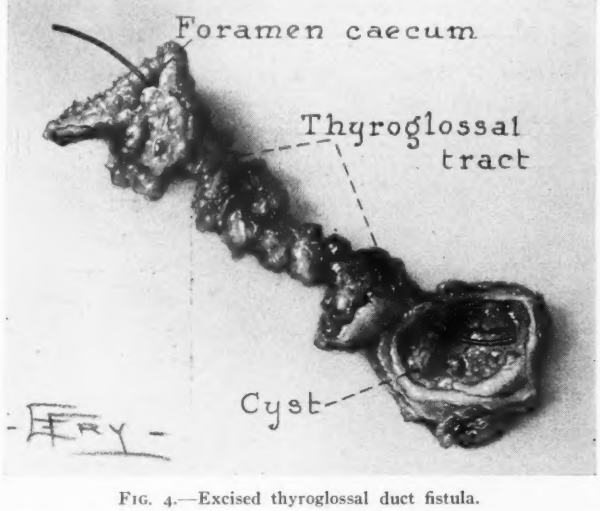

FIG. 4.—Excised thyroglossal duct fistula.

inset). A small rubber tube drain is placed deep in the muscles of the tongue and the skin wound is closed with interrupted dermal sutures.

From the years 1920 to 1938, inclusive, this operation has been performed at the Mayo Clinic in 293 consecutive cases. The sex incidence of the

patients was two males to one female. Operation was performed upon only 32 patients who were less than ten years of age. One hundred forty-four patients were between the ages of ten and 40; 90 were between 30 and 50 years, and only 27 beyond the age of 60 when the Sistrunk operation was performed at the clinic. The cyst or sinus had been noticeably present in some patients since birth, whereas in others it was present from a few weeks to even years. A summary of data on the age at time of operation at the clinic and the sex of the 293 patients is given in Table I.

TABLE I

AGE AND SEX

Sex	Number	Per Cent	Age in Years		
			Mean	Youngest	Oldest
Males	194	66.2	28.4	3	66
Females	99	33.8	25.1	4	58
Total	293	100.0	27.3	3	66

We were able to definitely trace as to recurrences, either by means of a questionnaire or by examination, 261 (89.1 per cent) of the 293 cases. In 166 (63.6 per cent) of these 261 cases, operations, usually multiple, had been performed prior to the patient's admission. Several of these patients had had from three to ten operations. In 41 of them the operative procedure was limited to incision and drainage, whereas in 125 at least one previous attempt had been made to excise the thyroglossal tract. In addition, in some instances the duct had been curetted or cauterized with electric cautery, carbolic acid, and other caustics. Frequently surgical therapy was supplemented by either roentgenotherapy, radium or both. In several cases ultraviolet irradiation, diathermy, and iodine both externally and internally had been administered. The operation in this group of patients presented considerable technical difficulty. Data on the recurrence of the sinus, cyst or fistula in the 261 traced cases are given in Table II.

TABLE II

RECURRENCE FOLLOWING OPERATION

	Total Cases *	Recurrences	
		Cases	Per Cent
Previous surgical measures	166	3	1.81
No previous surgical measures	95	1	1.05
Total	261	4	1.5

* Only 261 cases (89.1 per cent of the total 293 cases) were definitely traced as to recurrences included in this table.

There were only four recurrences in the entire group of 261 traced cases. The only patient who had recurrence in the group who had not had a previous operation was a man, 43 years of age. He had noted the thyroglossal duct cyst during his youth and was one of the first patients operated upon, using this method in 1920. Shortly after the operation, recurrence was noted.

The three patients who had undergone surgical procedures prior to the

Sistrunk operation performed at the clinic and had recurrence are interesting. The cyst of one patient, a man aged 31 years, had been noticed when he was two years old. It had been incised on many occasions, cauterized with carbolic acid on others, and a radical surgical excision attempted on two others. When the Sistrunk operation was performed at the clinic, the enormous amount of scarring and resulting deformity made a satisfactory operation impossible. Recurrence took place a few months after this operation. We recently have operated upon this patient again and, we feel, with much greater success. The second patient, a man aged 34 years, had noticed the cyst at the age of 17. This was incised several times and two attempts at radical removal were made elsewhere. The recurrence took place shortly after the operation performed at the clinic. The third patient, a youth of 18 years, had the cyst at the age of 19 months. It had been incised on several occasions and three attempts at radical removal had been made. When we first saw the patient the cyst was acutely inflamed, so it was opened and drained, and a few days later, in spite of the fact that the inflammation had not subsided, an attempt at radical removal was made. The tissues were so fragile and inflamed that the operation was exceedingly difficult. Recurrence took place within a few weeks following the operation. In the last two cases recent operation for these recurrences has been performed successfully.

COMMENT.—The effectiveness of the operation as proposed by Sistrunk for the complete removal of a thyroglossal duct cyst, sinus, or fistula is well established by the aforementioned results.[4, 5] Recurrences for the most part can be attributed to the presence of extensive scarring and various degrees of infection as a result of previous surgery. We feel that one of the recurrences in our series could possibly have been avoided and that recurrences in general can be avoided if the radical operation is postponed until the presence of acute infection has subsided. In cases in which there is considerable surrounding inflammation, it is usually best to make a simple incision and then several weeks after the inflammation has subsided completely perform the radical operation. Certainly, a thyroglossal duct cyst should not be incised unless inflammation is present for an unopened cyst makes the operation technically less difficult. When incision and drainage is performed, it should be undertaken only with the understanding that later radical excision will be necessary for cure. We have found, as Sistrunk has pointed out, that the injection of some dye, such as methylene blue, facilitates the tracing of the tract and aids in the detection of lateral branches in cases in which the cyst either has ruptured or has been previously treated surgically. In a few instances in which the cyst and its attachment have ended at the hyoid bone, because of a complete obliteration of the portion of the tract between the hyoid bone and the tongue the dissection was not carried through the muscle of the tongue. Thus the opening into the mouth with its resultant discomfort for the patient was avoided. In none of these instances has there been any evidence of recurrence. We realize that a sufficiently long period has not elapsed in all cases to exclude all possibility of a recurrence, but it has been our experience that recurrences which are due to incomplete removal of the

tract usually take place relatively soon after operation. In a few isolated instances they have occurred a few years after operation, but these have been exceptionally rare and for this reason we feel reasonably certain in our analysis of these cases. Only cases which were checked carefully either by a follow-up letter or by recent examination at the clinic were included in the study.

SUMMARY

A series of 293 cases of thyroglossal duct cyst in which the Sistrunk operation was employed were reviewed. Two hundred sixty-one of these cases were satisfactorily traced as to the possibility of recurrence. There were four recurrences in this series. The recurrences for the most part can be attributed to the presence of extensive scarring and infection as a result of previous surgery. The effectiveness of the Sistrunk operation stands out in sharp contrast with the many failures common to the less radical procedures.

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DISCUSSION.—DR. FRANK H. LAHEY (Boston, Mass.): I have been much interested in this very valuable paper. I think we must all agree that it was not until Doctor Sistrunk called attention to the necessity of removing the central section of the hyoid, excising the thyroglossal tract up to the tongue, that we began to get good results in cases of thyroglossal cysts. Probably we have all had the same experience when we first performed the operation. The percentage of recurrences was high, but since performing this radical operation the percentage of recurrences has been zero. We have operated upon about 200 patients with similar results.

There are a few things to be said regarding the operation: The tract cannot be traced above the hyoid in most cases. In certain cases there are collar-button tracts above and below and the tract can be well brought out. A central block of tissue must be taken out up to the base of the tongue. One does not have to be warned about the disadvantage of the longitudinal incision. One has only to see such an incision to know how terrible it is. It makes a checkrein running down the chin to the hyoid which is extremely difficult if not impossible to correct. I think one must be careful in excising the central portion of the hyoid bone to get an adequately wide removal. We were, in the beginning, disturbed because we could not resuture the hyoid, but this is not necessary. In a great many cases no attempt is made to do anything but suture the muscle.

As Doctor Pemberton has said, the thyroglossal tract represents the course along which the thyroid gland descends from its origin at the base of the tongue. Occasionally the thyroid gland does not descend and one then has what is called a lingual thyroid. We have had three patients with this condition in which all of the thyroid gland occurred as a mass of tissue at the

base of the tongue and so interfered with swallowing, as it continued to grow in size, that it required removal. Our experience with these lingual thyroids, related as they are to the descent or nondescent of the thyroid, may be of interest. We have never attempted to remove one of these lingual thyroids through the neck. I think it is the wrong way. In all three of these cases the lingual thyroids have been successfully removed through the mouth. One must remember also that probably all these patients will be myxedematous after removal of the lingual thyroids since they represent all of the thyroid present. The mouth is widely dilated and silk traction stitches are placed on each side of the tongue. It is extremely important that these stitches be inserted well back in the tongue so that the tongue can be pulled out to the forward portion of the mouth where the lingual thyroid can be dealt with under direct vision. I undertook the first one of these cases with considerable trepidation. I feared that the bleeding would not be easy to control. The dissection of the thyroid at the root of the tongue can be done very satisfactorily by splitting the tongue. The mass can be removed without difficulty, bleeding can be controlled and when all the vessels have been ligated, the tongue can be quite accurately approximated. Lingual thyroids are quite rare but one should have in mind their possible presence if a tumor is found on the back of the tongue and in the region of the foramen cecum.

Dr. Harold L. Foss (Danville, Pa.): It was not completely clear to me, from Doctor Pemberton's paper and Doctor Lahey's discussion, whether, routinely, the hyoid is divided by them or, especially, in what percentage of cases a segment of hyoid is excised. We have had several recurrences in my own clinic even with resection of the hyoid bone. Of course, a most complete excision of the entire tract must be carried out, as was pointed out by Sistrunk and to-day emphasized by Doctor Pemberton. It has been many years since I assisted the late Dr. Walter Sistrunk, and years since I read his original paper. In all probability he advised, for completeness' sake, removing a segment of the hyoid along with the entire fistulous tract, but I have forgotten. Doctor Pemberton will enlighten us, I am sure.

Dr. Vilray P. Blair (St. Louis, Mo.): In regard to the points made by Doctor Lahey, I once made a total excision of a lingual thyroid gland and decided that in future I would remove but part of the protrusion and save the necessity of subsequent thyroid gland extract treatment.

Dr. J. DeJ. Pemberton (Rochester, Minn., in closing): In regard to the lingual thyroid, I want to say that I think Doctor Lahey has brought out a very important point. The success of the operation on lingual thyroid is dependent on adequate exposure and this can be obtained best by means of traction sutures on the tongue. If intratracheal anesthesia is used, the operation is not difficult, since the bleeding can be controlled by suturing.

Doctor Foss asked if we removed a section of the hyoid bone routinely. We do; since at this point the thyroglossal duct cannot be visualized, in order to be sure that complete dissection is carried out, the central segment of the hyoid bone must be removed, as the duct sometimes runs through the bone itself. I think that we owe a great deal to Doctor Sistrunk for his efforts in standardizing this operation. As you know, up to his time, the medical profession looked on these sinuses in the light of a surgical rat hole, that is, they were annoying and troublesome to the patient but they were not dangerous to health. Therefore, no one took pains to devise a satisfactory operation for their removal.

RESULTS OF FACIO-HYPOGLOSSAL ANASTOMOSIS IN THE TREATMENT OF FACIAL PARALYSIS*

CLAUDE C. COLEMAN, M.D.

RICHMOND, VA.

FROM THE DEPARTMENT OF NEUROLOGICAL SURGERY, MEDICAL COLLEGE OF VIRGINIA, RICHMOND, VA.

SURGERY of the facial nerve may be required for the restoration of function when the nerve is paralyzed from trauma or disease[1] and to reduce or abolish function when the muscles it supplies are involved in severe spasm. In a paper[2] before this Association in 1936, attention was called to the paroxysmal disturbance of function characteristic of surgical diseases of the cranial nerves. In addition to the facial, other cranial nerves showing explosive disturbance of function are the fifth and ninth in intractable neuralgia, the eighth in Ménière's disease, and the eleventh in spasmodic torticollis. Destructive operation upon the affected nerve is required for the relief of these conditions. Operation upon the facial nerve is resorted to in a large majority of cases to restore function, and in this respect surgery of the facial nerve differs from that of other cranial nerves in which the operation is always destructive.

The deformity caused by severe facial paralysis is too familiar to require description but an analysis of the components of this deformity may be of some interest. The most conspicuous and embarrassing feature of the deformity of unilateral facial paralysis arises from displacement of the mouth by unopposed contraction of the muscles of the healthy side, whereas the alteration of facial appearance caused by paralysis of other muscle groups, such as those of the upper lip, eyelids and brow, is not exaggerated by the activity of the muscles on the normal side (Fig. 1). From loss of function of the mandibular branch of the nerve there is not only sagging and deviation of the lips when the face is in repose but emotional expression on the healthy side draws the mouth out of alinement and suddenly produces a marked increase in the deformity. From observation of patients upon whom section of the upper branches of the nerve had been performed for the relief of facial spasm or in cases of accidental division of these branches, we have been greatly impressed with the slight facial deformity resulting from loss of action in the muscles of the upper part of the face when balance of the mouth is retained by action of the lower branch of the nerve (Figs. 2 and 3). There may be sagging of the lower eyelid from loss of innervation to the orbicularis oculi but this is not a conspicuous deformity and may be greatly improved by external canthoplasty.

Decompression of the nerve in the bony canal, direct suture and nerve

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graft have been enthusiastically advocated for lesions in or peripheral to the bony canal and these reconstructive operations will probably supersede anastomosis when the location of the lesion is favorable for such procedures.[3] In my observation of the results of suture and graft of the trunk of the facial nerve I have been impressed with the failure of these operations to restore emotional expression or to prevent mass movements of the face. The development of mass movements of the face and the permanent loss of normal emotional expression after nerve graft or suture are due to defective regeneration of the nerve. Following severe paralysis, regeneration of the facial nerve, whether spontaneous or aided by suture or graft, proceeds with some disorder of the regenerating fibers and with deflection of the new fibers from the course they normally follow. Ford and Woodhall,[4] in a very interesting paper, have discussed the effects of misdirection of regenerating nerve fibers upon the subsequent action of the facial muscles. After section or a severe degenerative lesion of the nerve, new fibers from that part of the nerve normally intended for one group of muscles may penetrate every muscle group within the facial domain, thus preventing the isolated action of individual muscle groups so essential to emotional expression. The result is that when the patient attempts to close the eye, the muscles about the mouth contract and efforts at expression by action of the muscles about the mouth are accompanied by associated contraction of the orbicularis oculi. While it is possible in operation for direct suture to preserve the nerve pattern in a gross way, straying and deflection of the new fibers cannot be prevented. This phenomenon of faulty regeneration fully explains the failure of any surgical procedure to restore normal movements of the face and emotional expression after severe lesions of the trunk of the nerve. The situation is altogether different after successful repair of one or more peripheral branches of the facial nerve. In such cases the misdirection of fibers of the regenerating nerve would affect only the group of muscles normally supplied by that branch of the nerve and would not mar facial ex-

FIG. 1.—Facial paralysis with marked atrophy and deformity following complete removal of a left acoustic neuroma two years previously. Patient's general and neurologic condition excellent with exception of facial paralysis. Typical deformity of complete facial paralysis when atrophy of the muscles has taken place. No response to galvanic stimulation on affected side. Anastomosis not indicated. Operation for support of the face by fascial strips after Brown's method, November 9, 1939, with immediate improvement in patient's appearance. Operation too recent for postoperative photographs. Photograph used to illustrate severe progressive deformity when innervation of facial muscles is permanently destroyed.

pression. The results of suture of important branches of the nerve are highly successful from every standpoint.

While several surgical procedures are in common use for the relief of deformity resulting from severe facial paralysis, anastomosis of the facial with another motor cranial nerve is the only procedure which will restore innervation to the paralyzed facial muscles following an intracranial lesion of

FIG. 2.—Paralysis of the upper branches of the facial nerve caused by a glass wound in the parotid region. As is usual in such cases, the mandibular branch of the nerve escaped injury. Photograph illustrates the important part played by the mandibular branch of the nerve in the preservation of facial symmetry. Even in smiling, the mouth is fairly well balanced although there is slight droop of the upper lip. Some widening of the palpebral fissure is shown. Suture of the individual branches of the nerve, November 2, 1939, two months after injury. Evidence of returning function expected in about three and one-half months after operation.

FIG. 3.—Patient shown in Fig. 2, showing inability to close eye on the affected side. If it is impossible to repair the branches of the nerve to the muscles about the eye, considerable improvement may be obtained from canthoplasty.

the nerve. There can be no adequate substitute for anastomosis in such cases. Intracranial injuries of the facial nerve formerly were rare but now are frequently seen due to the increasing number of operations for cerebellopontine angle tumor, with destruction of the facial nerve by complete removal of the tumor and its capsule (Figs. 4, 5 and 6).

When anastomosis is required for innervation of the paralyzed facial musculature we have preferred the hypoglossal to the spinal accessory because of its functional similarity to the facial. Facio-hypoglossal anastomosis will restore movement to the paralyzed muscles in practically every case, will

balance the face in repose and prevent atrophy of the muscles on the affected side. These results fully justify this type of surgical procedure for the improvement of a deformity which without operation becomes progressively worse (Figs. 7, 8, 9 and 10). Function of the muscle groups cannot be individualized after facio-hypoglossal anastomosis, and when the patient responds to emotional stimuli the expression of emotion is registered only on the healthy side. Smiling is accompanied by slight deviation of the mouth to the normal side due to delayed action and weakness of the muscles about the mouth on the affected side. Frontalis action has been recovered in only one case in our series after hypoglossal anastomosis. The loquacious patient is the most unfavorable type for facio-hypoglossal anastomosis, and in such patients the spinal accessory might be used. There is a marked difference in the activity of patients' tongues, not only with respect to talking but also when swallowing. Associated movements of the face are produced by action of the tongue after hypoglossal anastomosis. In some patients in whom the movements of the tongue during swallowing or talking are very active, there are exaggerated contractions of the muscles about the eye and lips, giving the patient in an extreme case a grotesque expression. Success of anastomosis operations depends to a great extent upon the patient himself.

FIG. 4.—Complete left facial paralysis following total removal of an acoustic neuroma. There was also injury to the left trigeminal nerve. The lids have been sutured to protect the eye. Left facio-hypoglossal anastomosis, March 8, 1937, to relieve the paralysis.

Results of facio-hypoglossal anastomosis compare favorably with those of suture or graft of the nerve trunk, but anastomosis requires the sacrifice of another cranial nerve and if the hypoglossal is used it necessitates control of the movements of the tongue to minimize associated movements. Atrophy of the tongue caused by section of the hypoglossal appears to be of no important significance and causes no appreciable interference with speech or deglutition (Figs. 11, 12 and 13).

Much has been said about reeducation of the patient who recovers function of the muscles following operation for facial paralysis. It is difficult to understand how such reeducation could be very successful when directed only to improvement of action of the previously paralyzed muscles. Accepting the experimental evidence of regeneration of the nerve and the phenomena of deflection of axis cylinders from normal pathways, one can readily appreciate

the obstacles to reeducation. With misdirection and deflection of regenerating axis cylinders the entire facial musculature on the affected side is made a single functional unit and the facial muscles move *en masse*. It seems impossible for reeducation, under such circumstances, to effect materially a dissociation of action of various muscle groups. In some cases efforts at reeducation may result in increasing the patient's deformity by bringing about overactivity of the affected side, causing grimaces which may become habitual. We believe that much more can be accomplished in improving the results of operation by instructing the patient to suppress facial movements and

FIG. 5.—Photograph of the patient in Figure 4, 21 months after anastomosis. Recovery of motion in all muscle groups except the frontalis and platysma. No deformity is evident when the face is in repose.

FIG. 6.—Photograph of the patient shown in Figures 4 and 5, shown closing the eyes with little associated movement. Atrophy of the muscles of mastication caused by injury to the trigeminal nerve when the tumor was removed.

emotional expression on the normal side. Suppression of facial movements should become a fixed habit.

The residual effects of facial paralysis after maximum recovery of function following anastomosis operations, obviously, will vary in importance according to the patient's occupation, age, sex and social position. One must give serious consideration to the unavoidable destruction of the facial nerve in operations for the complete removal of acoustic tumors. The patient should be fully informed before operation is undertaken that complete removal of an angle tumor is almost inevitably followed by facial paralysis and he is entitled to know in detail the features of such paralysis. In only one case have we been able to completely remove an acoustic neuroma without destroying the facial nerve (Figs. 14 and 15). Facial paralysis is a small sacrifice to make

for the assurance of permanent eradication of an acoustic neuroma as a primary procedure especially when one reflects that operations for recurrence of these tumors following incomplete removal are highly dangerous and may be impossible. The argument for a more conservative type of operation for acoustic neuroma is strengthened in certain cases by the fact that the tumor is a benign one, and after complete enucleation of the tumor with almost complete removal of the capsule, recurrence probably would not take place for a great many years.

FIG. 7.—Results of facio-hypoglossal anastomosis for relief of facial paralysis following complete removal of a left acoustic neuroma. Tumor symptoms completely relieved by operation and the patient is actively engaged in his work. Photograph shows the face in repose. The mouth is well balanced, there is little difference in the width of the palpebral fissures and very slight alteration of the patient's facial appearance. Photograph two years after anastomosis.

FIG. 8.—Photograph of the patient in Figure 7, showing limitation of emotional expression with some weakness of the left side of the mouth. The functional results of the operation in this case were highly satisfactory.

When there is residual paralysis or incomplete recovery of important muscle groups the patient's appearance may be improved by supporting the face with fascial strips, as described by Brown,[5] in 1938; and stabilizing the paralyzed side in this manner is the only operation that will improve the deformity when the facial muscles have lost galvanic response or when there is extensive destruction of peripheral branches of the nerve. In many cases the combination of nerve anastomosis and fascial support gives the best results, and we believe this combination of surgical procedures should be employed more frequently.

Facio-hypoglossal anastomosis is an operation of little difficulty and negligible risk. An incision is made extending from the tip of the mastoid process downward, following a crease in the neck about 2 cm. beneath the mandible, extending in the direction of the thyroid cartilage (Fig. 16). The hypoglossal nerve is first isolated at the level of the cornu of the hyoid bone and traced backward to where it curves around the occipital artery, but it is not sectioned until the facial trunk has been identified and divided. Exposure of the facial nerve in the digastric fossa is accomplished very easily by retracting

FIG. 9.—Photograph of the patient in Figures 7 and 8, showing mass movements of the face on the left side when efforts are made to forcibly retract the angle of the mouth on that side. No evidence of frontalis function.

FIG. 10.—Photograph of the patient in Figures 7, 8 and 9, showing closing of the eyes, which is accompanied by movements of the muscles about the mouth. Photograph one year after anastomosis.

the digastric muscle downward and following the digastric branch of the facial upward to the trunk. Section of the facial trunk is made as close as possible to the stylomastoid foramen. The branches to both the stylohyoid and the digastric muscles are sacrificed but the resulting paralysis is apparently not of great importance, although it may account for some difficulty in swallowing. After division of the facial the peripheral stump is pulled downward and placed in a suitable position for suture. The hypoglossal is then sectioned sufficiently close to the tongue to permit the central segment to be approximated with the trunk of the facial without tension (Fig. 17). This requires section of the thyrohyoid branch of the hypoglossal and in some cases it is impossible to approximate the ends of the two nerves satisfactorily without freeing the descendens hypoglossi from the main trunk of the nerve. Anas-

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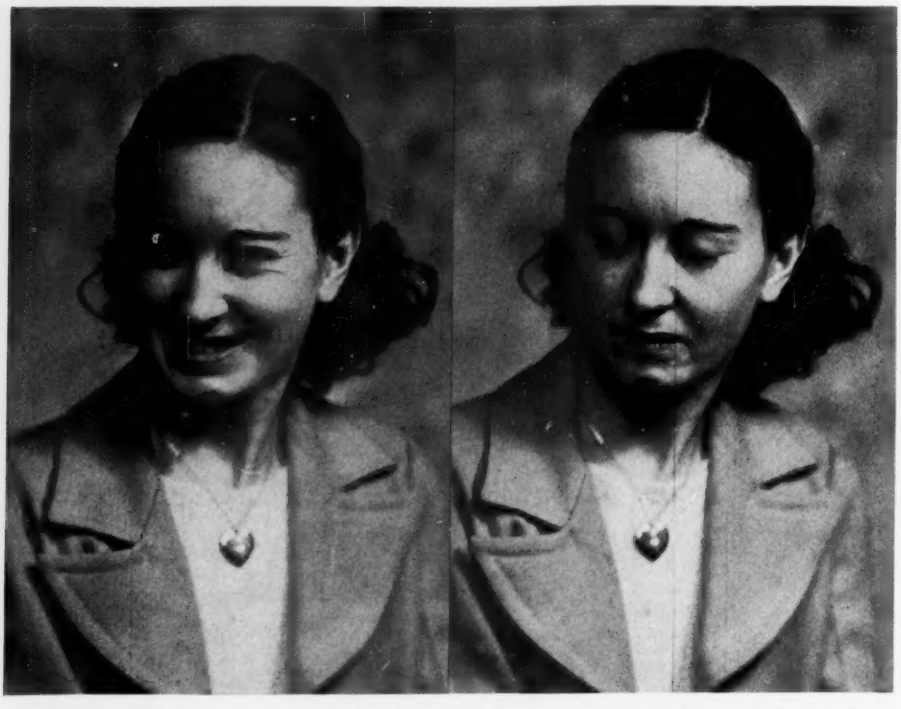
FIG. 11.—Marked facial paralysis produced by section of the nerve for the cure of left facial spasm. Section with immediate suture of the upper branches of the nerve had produced temporary relief of the spasm with recurrence. Facio-hypoglossal anastomosis for relief of the paralysis.

FIG. 12.—Photograph of the patient shown in Figure 11, two and one-half years after facio-hypoglossal anastomosis. Very little deformity with face in repose. There was no return of function of the frontalis muscle but the corrugator on the affected side is active.

FIG. 13.—Photograph of the patient in Figures 11 and 12, shown attempting to smile. There is some lagging of the angle of the mouth on the left side and slight widening of the palpebral fissure. The patient's facial expression was normally vivacious and good effects of the operation were somewhat marred by associated movements, particularly those about the eye.

tomosis should be performed carefully, with accurate approximation of the nerve sheath and without projection of nerve fibers between the sutures. Three arterial sutures are sufficient to unite the nerves. In some cases we have sutured the proximal end of the descendens hypoglossi to the peripheral end of the hypoglossal trunk. Atrophy of the tongue following this procedure has developed in every case except one, showing that attempts to preserve the

FIG. 14. FIG. 15.



FIGS. 14 and 15.—Photographs of the patient after recovery from facial paralysis caused by removal of a large left acoustic tumor and its capsule. The intracranial portion of the nerve was greatly lengthened and it was protected during the operation. There was a temporary facial paralysis lasting about six months. The patient was bedridden for some time before operation and was unconscious when admitted to the hospital. She made a complete recovery from tumor symptoms with the resumption of full duties as a school teacher. Photograph three years after removal of tumor. No evidence of impaired facial expression.

trophic supply to the tongue are generally futile. The junction of the anastomosed nerves should be covered by the digastric muscle which is retracted downward during the operation.

The first evidence of returning function of the nerve following anastomosis is improved tone in the paralyzed muscles and the restoration of balance of the mouth. In about three and one-half months feeble movements appear about the angle of the mouth and spread upward to the eye muscles which show definite contraction in about six months. During the stage of paralysis the sagging face should be supported by adhesive strips and the muscle tone preserved as much as possible by facial massage. The maximum improvement is reached after about two years.

Regardless of the type of surgical procedure employed for the relief of complete facial paralysis, one must be impressed with the fact that the maximum recovery following all operations leaves much to be desired, and that the patient's facial appearance is never restored to normal. Facio-hypoglossal anastomosis for the relief of paralysis following intracranial lesions of the nerve will balance the face in repose, restore movement of the paralyzed muscles, and prevent further disfigurement resulting from atrophy of the muscles.

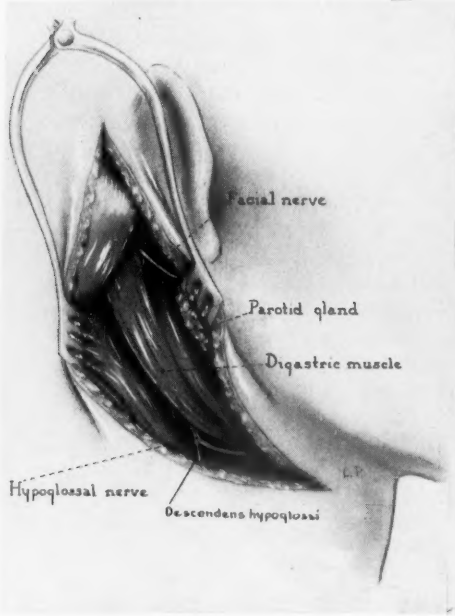


FIG. 16.—Drawing showing exposure for right facio-hypoglossal anastomosis. The sternomastoid muscle is retracted backward. The hypoglossal and the facial nerves are shown. By retracting the digastric muscle downward and backward, a small branch of the facial nerve to this muscle can usually be found. Tracing this branch backward leads to the trunk of the nerve.

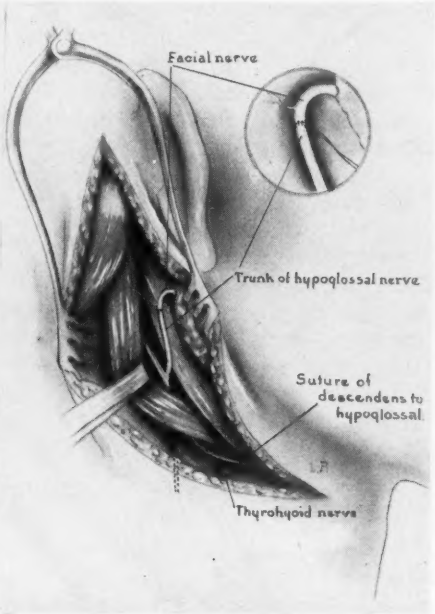


FIG. 17.—Drawing showing the hypoglossal trunk divided and anastomosed to the peripheral trunk of the facial. The thyrohyoid branch is sacrificed in mobilizing the nerve for suture. The dotted lines show the course of the descendens hypoglossi which has been sutured into the peripheral segment of the hypoglossal nerve. (Redrawn from Facial Spasm. ANNALS OF SURGERY, May, 1937.)

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DISCUSSION.—DR. CHARLES BAGLEY, JR. (Baltimore, Md.): Doctor Coleman has presented to us, under the title of "Facial Nerve Surgery," a very interesting group of patients after removal of acoustic nerve tumors. He has done this in his usual modest fashion, with no effort to call attention to the difficulties encountered in the removal of this type of tumor further than to mention the necessary destruction of the facial nerve in the majority of cases. Any tumor in the cerebellar pontine angle is rather inaccessible, so that the operation presents many mechanical difficulties. The actual incorporation of the seventh and eighth nerves in the tumor mass forces one to choose between a subtotal removal or total loss of function of the seventh and eighth nerves on the side of the tumor. In theory, this would seem to be a very simple choice. On the contrary, one always dreads to face a patient whose complete facial paralysis is of his making. Doctor Coleman has, in these cases, attempted to give the patient the advantage of total removal and then to restore the function of the facial nerve.

In this type of paralysis, where one knows the nerve has been destroyed, waiting for spontaneous return is not necessary. Doctor Coleman's plan of repairing the nerve at the earliest possible moment has the advantage of restoring the nerve before damaging atrophy of tissue has advanced too far. The functional results shown by his patients are quite satisfactory at the present time, and, as some of these operations have been performed rather recently, there will be greater improvement as time goes on.

Doctor Coleman is to be congratulated on the very excellent results obtained in a number of patients with complete removal of acoustic nerve tumors in whom he has been able to reestablish facial nerve function so that the price which they have paid for complete removal will ultimately be minimal.

DR. JOSEPH E. J. KING (New York, N. Y.): I want to congratulate Doctor Coleman on his splendid paper, his management of facial nerve injuries in general, and his fine results in this series. I have always felt that facial nerve injuries should be considered under three headings: First, those in which the nerve is destroyed in its intracranial course; second, that group in which the nerve is injured or destroyed in its course through the temporal bone; and third, those where the injury has occurred in the peripheral portion of the nerve. Therefore, the operation of choice for restoration of the nerve will depend upon the level or point where the nerve is destroyed.

Most of the cases which have come under my observation were those in which the injury was associated with mastoid disease or following mastoidectomy. All of these I have referred to Dr. Thomas Tickle for nerve graft. I have done this for three reasons—he has obtained very good results with his cases, there is no scar on the neck, and there is no associated hemiparalysis and atrophy of the tongue.

In those cases in which intracranial destruction has taken place, a graft is out of the question, and in such cases the anastomosis described by Doctor Coleman is naturally the procedure of choice. In cases in which the damage to the nerve is in the peripheral portion, and suture cannot be accomplished, one must resort to fascial strips.

DR. FRANK H. LAHEY (Boston, Mass.): I am sure that this is no place for the general surgeon to enter into a discussion of nerve anastomosis—in our clinic this is the field of the neurosurgeon. On the other hand, I think if we have differences of opinion regarding choice of nerves, we should state them. I have had many opportunities of viewing end-results following injury to hypoglossal nerves, and I am impressed with this as an undesirable situation.

Doctors Poppen and Horrax feel the same way, and, therefore, I may say we prefer to use the spinal accessory nerve. Doctor Poppen has performed most of these operations and, although the functional result has been excellent, the spinal accessory nerve has been chosen in 19 cases and the hypoglossal in one. I have had several cases in which the hypoglossal nerve has been involved in malignancy and its sacrifice has been required. It must be that there is a variation in the degree of dysfunction following paralysis of the hypoglossal nerve. Certainly, in some of my surgical cases I have thought it undesirable to have patients talk with a thick speech and to have difficulty with mastication. You have to question yourself—will you choose to sacrifice the function which has to do with speech and swallowing, or the one which has to do with the last 90 degrees of abduction of the arm; which is the function taken away when the spinal accessory is used in the anastomosis? The last 90 degrees of motion is accomplished by scapular rotation through the accessory innervated trapezius. We have felt that it is better for the patient to lose this last 90 degrees of arm abduction than to have difficulty with speech and swallowing. That may be only a difference of opinion but it has been the conclusion of Doctors Horrax, Poppen and myself.

Dr. Vilray P. Blair (St. Louis, Mo.): The most striking feature of this contribution is Doctor Coleman's analysis of the functional results of nerve anastomosis—and that might have something to do with the choice of the repair method. Whether we like the fascial strips or not, most of ours are performed in that way because most of the cases we see are either too old to offer hope of restoring muscle action or the nerve injury has been of small filaments in the parotid gland, and it is practically impossible to make an anastomosis in these. If the fascial strip operation is performed carefully, an almost perfect restoration of the quiescent face will result. Many have tried to obtain muscular motion by turning down a piece of the masseter muscle, but I have never been sufficiently impressed with the procedure to try it. Brown's real contribution was not trying to turn down a piece of muscle which might slough or sclerose but his procedure is to extend the fascia far enough to allow attachment to the undisturbed muscle, which is, I think, the difference between success and probable failure.

Dr. Claude C. Coleman (Richmond, Va.) closing: I want to thank Doctors Bagley, King, Blair, and Lahey for their part in the discussion. I agree with Doctor King that nerve graft may be used as a substitute for anastomosis whenever the lesion is favorably situated. Nerve grafts for lesions in the facial canal will not be followed by associated movements but they do not prevent mass movements, and I have never seen a nerve graft restore emotional expression.

In facio-hypoglossal anastomosis, movements of the face are initiated by moving the tongue. Unless action of the tongue is repressed, associated movements of the muscles about the eye may be troublesome. Anastomosis operations, of course, are much simpler than nerve grafts in the facial canal. A thorough knowledge of the surgical anatomy of the temporal bone is essential to exposure of lesions of the facial nerve in the facial canal.

Doctor Lahey has expressed a preference for the spinal accessory nerve when a cranial nerve must be used to restore innervation to paralyzed muscles of the face. The choice of the nerve to be used has long been a subject of much controversy. As stated in my paper, we have used the hypoglossal because of its closer functional similarity to the facial. However, there are cases in which the spinal accessory may be preferred. We have used the

spinal accessory in only one case, that of an old lady with facial paralysis following complete removal of an acoustic neuroma, who objected to any operation which would affect the nerve of her tongue. The point not being of great importance in her case, we were willing to use the spinal accessory.

From a careful study of patients, both before and after facio-hypoglossal anastomosis, I have not been impressed with any loss of function from sacrifice of the hypoglossal nerve. Voice records have been made of these patients before and after operation, showing no noticeable interference with speech. In complete lesions of the nerve proximal to its exit from the facial canal, function of the digastric, stylohyoid and buccinator is lost. That of the digastric and stylohyoid would be permanently lost regardless of which nerve is used in the anastomosis. It may be that some impairment of the hyoid stabilization has something to do with the speech and it is very probable, too, that the buccinator, like the frontalis, does not recover as completely after nerve graft or anastomosis as other muscle groups. Impairment of function of speech or deglutition, attributed to section of the hypoglossal on one side, might be due in part at least to impairment of function in these three muscles and not to loss of the hypoglossal.

From my observation of section of the spinal accessory as a part of the surgical treatment of spasmodic torticollis, I am inclined to believe that the loss of the hypoglossal is of less importance than that of the spinal accessory. In view of the fact that section of either of these nerves carries no considerable penalty, it would seem that the choice between the hypoglossal and the spinal accessory nerves in anastomosis should not be determined by the loss of function incident to section of the substituting nerve but should depend upon which nerve would bring about better facial movement.

THE FREQUENCY AND DIAGNOSIS OF HIATAL HERNIA*

DONALD GUTHRIE, M.D.,

AND

F. HARRIMAN JONES, M.D.

SAYRE, PA.

HIATAL HERNIA is the term used to designate a herniation of abdominal contents through the esophageal hiatus of the diaphragm. All herniae of the diaphragm, therefore, do not come under this heading, but of recent years the frequency with which the hiatal herniae have been recognized has been so great that the attention of the medical profession has been called to this condition in a startling way.

The first herniae of the diaphragm that were recognized were of the traumatic type. Ambroïse Paré reported two cases of the traumatic type as early as 1610 and, in 1769, Morgagni wrote the first monograph on the subject. However, until recent years the hiatal type has not been well recognized. At the Mayo Clinic the first hiatal hernia was found at operation in 1908, but it was not until 1921 that the diagnosis was made by roentgenographic examination.

Moersch,[4] in going over the records at the Mayo Clinic, found that, from 1932 to 1937, there were 267 cases in which there was a positive diagnosis of diaphragmatic hernia made at roentgenographic examination. Of this number, there were 246 of the hiatal type in contrast to 15 of the traumatic and six congenital herniae.

Harrington[3] has gone back in the Mayo Clinic records to 1908, when the first case of hiatal hernia was recognized, as mentioned above, and found that from that time until 1926 the diagnosis of hiatal hernia was made just 17 times, but that from 1926 to 1938 the diagnosis was made 399 times. This is 23 times as many cases in the past 12 years as in the previous 18 years. Also, a marked increase in the roentgenologic diagnosis is startlingly brought out by the contrasting figures of one case in 1921 and 99 cases in 1937. During the period 1908–1938, a diagnosis of diaphragmatic hernia (all types) was made roentgenologically, or at operation, in 514 cases.

Others are also noting the same changes in the frequency of the diagnosis of this condition. At the Guthrie Clinic[2] there were five cases of hiatal hernia recognized and operated upon from June, 1927, to January, 1938. Since then (until November, 1939) there have been 14 cases found. These will be considered later in the paper.

Harrington believes that the hiatal hernia is no more frequent now than 20 years ago but that the apparent increase in the number of cases is due to the fact that both clinicians and radiologists are looking for the condition and

* Read at the Fifty-second Annual Meeting of the Southern Surgical Association, Augusta, Ga., December 6, 7, 8, 1939.

both are coming to recognize the symptoms and findings of the disease. The diagnosis does not depend entirely on the radiologist but the clinician plays an important part by recognizing the symptoms and requesting the proper examination.

Hiatal herniae have been divided by Akerlund[1] into three types: (1) A congenitally short esophagus with partial or complete thoracic stomach; (2) a normal length esophagus not forming part of the hernia but with a para-esophageal hernia; and (3) a short esophagus forming part of the hernial contents. Moersch states that the first is usually due to embryologic retardation, but the possibility of such a picture occurring as a result of cicatrization of the esophagus cannot be dismissed. The other two types are of congenital origin in the same sense as inguinal herniae, and any factor that tends to increase intra-abdominal pressure may act as a contributory factor. Truesdale[7] believes the cause is a relaxation of the muscle structure of the diaphragm superimposed on a congenital deficiency of tissue surrounding the lower end of the esophagus. Moersch, Ude and Rigler,[8] and Akerlund all noted that the third or sliding type was more frequently found than the other two.

The first type needs little discussion. The esophagus may be so short that the stomach may lie almost entirely within the thorax. This is rare and is as a rule the only abdominal organ involved in the abnormal position. Usually the esophagus is not this short and only a portion of the stomach is above the level of the diaphragm. There is often a narrowing at the diaphragmatic level and frequently spasm or even ulcer formation with bleeding occurs at this point. It is not reducible under ordinary conditions. Harrington says it is not a true hernia but an abnormality of development. The second and third types of this classification are the only ones which he lists as true herniae.

In the second type, which has a normal length esophagus, it is found that the lower end of the esophagus is attached to the hiatal ring and does not form a part of the hernial contents which are para-esophageal. It is usually reducible and frequently reduced when the patient is in the upright position—a factor that may lead to a missed diagnosis if the patient is not examined roentgenologically in the supine position.

The third type, or sliding hernia, in reality has a normal length esophagus but one which has become shortened as the cardiac end of the stomach has pushed through the hiatus. The esophagus does not remain attached to the hiatal ring. Ordinarily it is also easily reducible and in some patients remains reduced when in the upright position. Either of these latter two types may become irreducible if adhesions form in the mediastinum holding the stomach above the diaphragm.

Many of the hiatal herniae produce few or no symptoms; if there are symptoms, they are usually not clear-cut or specific of the condition. In general, the amount and type of abdominal viscera herniated is in direct proportion to the symptoms; involving only a portion of the cardiac end of the

stomach may produce more severe symptoms than a larger hernia involving the greater part of the stomach. Harrington also points out that a large majority of the patients will already have had an average of three erroneous diagnoses made before the true nature of the disease is discovered. He lists these in order of frequency as cholecystitis, cholelithiasis, gastric ulcer, duodenal ulcer, hyperacidity, secondary anemia, cardiac disease, carcinoma of the cardia, stricture of the esophagus, appendicitis and intestinal obstruction. Morton[5] points out, however, that in older patients other diseases may actually be present and an accurate diagnosis becomes difficult.

One of the most constant and most frequent of symptoms is pain situated under the lower end of the sternum. This pain frequently radiates through to the back, is usually mild in onset, and comes on during or shortly after eating. Frequently, the patient will notice that liquids are easily taken but that solid food, unless very well masticated, will cause discomfort. Again, they will also note that after a very large meal they are more likely to have distress than if a small meal is taken and overeating is not indulged in. When present, this pain often leads to confusion with peptic ulcer because there may be relief from food, milk or soda. If the pain is more severe and associated with vomiting, one might easily confuse the diagnosis with gallbladder disease. Other symptoms are vomiting, hemorrhage, belching, weakness and anemia. Occasionally in the large herniae there is pulmonary or cardiac embarrassment with symptoms of dyspnea and palpitation. Spasm of the diaphragm may cause phrenic pain in the left shoulder or even simulate angina pectoris. Moersch points out that the symptoms frequently are intensified or aggravated by the recumbent position.

Bleeding comes from the mucous membrane of the stomach following erosion or ulceration and, when present, indicates an incarceration at the hiatal ring. Subacute perforation was demonstrated at operation in one of the cases reported by Guthrie and Brown.[2] Because of the heavy musculature of the stomach wall the stomach does not become strangulated. Harrington says that strangulation is not possible because of the "powerful musculature and rich blood supply of the gastric wall."

The definite diagnosis of hiatal herniae rests upon roentgenographic recognition. The hiatal hernia can be most easily seen with the patient in the recumbent position. Only in those where it does not reduce easily in the upright position or in those in which it is fixed by adhesions in the thorax can it be recognized while the patient is standing. In brief, the roentgenologic examination is carried out as follows: While standing in front of the fluoroscope the patient is asked to take one swallow of the barium mixture. This is watched as it passes down the esophagus and into the stomach. Often, as the barium passes the cardia there is a suspicion of a hiatal hernia. The routine examination of stomach and duodenum is then carried out as the patient finishes drinking the barium mixture. The table is then tilted so that the patient is in the prone position. Further examination of esophagus, stomach and duodenum are carried out. The patient is also turned face down so that

the barium filled portions of the gastro-intestinal tract may be studied from all angles. Roentgenograms are taken with the patient in the supine position. The head is turned to one side and a mouthful of barium is sucked up through a glass tube and swallowed. This barium is watched while it passes through the esophagus as the patient swallows against gravity. The exposure is made just after this part of the procedure.

In those having a congenitally short esophagus the hernia cannot be reduced and, therefore, is apparent in both upright and recumbent positions. The upper portion of the stomach is usually pointed and the esophagus empties into this upper or peaked portion. In the true hiatal herniae (the para-esophageal and sliding types) the contour of the herniated portion of the stomach is rounded. The distal end of the esophagus enters the stomach in its normal position. In the para-esophageal variety the lower end of the esophagus is fixed to the hiatal ring but in the sliding type it may be seen to change its relation in the thoracic cavity. It is important in this sliding type to determine whether the esophagus is of normal length or not.

Moersch stresses the value of esophagoscopy in the study of this condition. The findings in the patients with a congenitally short esophagus are a narrowing of the esophagogastric junction with a dilated portion below this point showing folds of gastric mucosa. There is no evidence of the normal diaphragmatic hiatus. The narrowing may be fusiform and from 1 to 7 cm. in length or may be a membrane which partially obstructs the lumen. There may be ulceration at this point which often simulates that of carcinoma.

The sliding type hernia may closely resemble the congenitally short esophagus. Here, however, the esophagus is more redundant and the point of narrowing is less pronounced and shorter. In some cases it may be advisable to examine the patient fluoroscopically with the esophagoscope in place, in order to determine the exact relation of the esophagogastric junction.

Esophagoscopy is especially indicated in the bleeding cases, in order to determine the extent of the erosion or ulceration and to rule out the possibility of another lesion causing the bleeding. Harrington feels that it should be carried out in every case of hiatal hernia in which operation is to be undertaken. Carcinoma of the cardia has been found in connection with hiatal hernia, and using esophagoscopy in making the positive diagnosis tends to minimize unforeseen therapeutic problems.

Not all hiatal herniae should be subjected to surgery, although in the larger herniae, and those producing severe symptoms, a surgical procedure is the only means to relieve the patient. An absence of symptoms or very mild symptoms are contraindications to surgery but it is important to have a record of the condition so that any increase in symptoms will not be judged to have a surgical or cardiac basis.

Root and Pritchett[6] believe that, in the medical management of these patients, they should be instructed to eat frequent, small meals and remain erect for some time after eating. They should not eat just before retiring at night. Moersch feels that the procedure of choice in patients with a con-

genitally short esophagus is to dilate the narrowing at the esophagogastric junction. He does point out, however, that, if there is no definite stenosis and no ulceration of the mucous membrane, it may not be a true congenitally short esophagus although the roentgenologic and esophagoscopic examinations might point to this. Harrington has operated upon such patients and found that the stomach could be reduced and that the esophagus was not actually shorter than normal. In the very obese patient a simple reduction in weight will often relieve the symptoms.

Harrington, who has had the largest experience in the treatment of hiatal herniae, divides them into three groups from the standpoint of treatment: The first includes those without or with only mild symptoms who do not need any treatment. In the second are those with moderate symptoms who require only conservative measurements, as regulation of diet and reduction of weight. In the third group are those with severe symptoms or those who have not found relief from the conservative means; these require an operative procedure. He also places in this operative group those patients with herniae larger than 5 cm. in diameter and all those where the colon may be involved in the hernia.

Interruption of the phrenic nerve should be effected before a radical operative procedure, and is a necessity if dealing with the congenitally short esophagus and thoracic stomach. In some cases, where a radical operation is contraindicated, section of the phrenic nerve may be employed as a palliative measure. When used with direct attack on the hernia, temporary interruption is preferred unless there is some indication to make it permanent. As a palliative means, it is employed to prevent spasm of the diaphragm and thus cut down the severe attacks with danger of incarceration of the stomach and to give the medical treatment a chance to heal ulcers of the gastric mucosa. It usually does not completely relieve the symptoms but the patients get along well if they are careful with their diet.

Operative Technic.—The abdominal approach is most frequently employed in repairing the hiatal hernia. By cutting the suspensory ligament to the left lobe of the liver it can be retracted to the right, exposing the left hypochondrium. The stomach should be explored for any pathologic lesion which may be associated with the hernia. Wide exposure is necessary and after being reduced from its thoracic position the stomach must be retracted strongly downward.

Harrington stresses the importance of removing the hernial sac. It is well to point out that the hernial sac in the hiatal hernia is in the posterior mediastinum and not in the pleural cavity. We pass a stomach tube after the sutures are placed to make sure the lumen of the esophagus is not constricted. The suture nearest the esophagus should include a small bite of the outer layers of the wall of this organ to insure its being fixed in normal position and to keep it from tending to draw again into the thorax. Before closing the abdomen, other organs, especially the gallbladder, should be explored.

Postoperatively, any signs of shock must be immediately combated. An

oxygen tent may be employed to advantage but we have not found many patients requiring this treatment. However, our series of operated cases is small. Ventilation of the lungs with carbon dioxide and oxygen twice a day for three days aids greatly in preventing postoperative pulmonary complications. Liquids may be started as tolerated by the patient, followed by a modified soft diet on the third or fourth postoperative day. The care of the wound is that of any other abdominal incision.

Ten of the 14 cases seen here since January, 1938, have been females. Nine of the 14 have been treated by conservative means. One was treated conservatively plus a dilatation of the esophagus. On one, a phrenectomy was the only operative procedure directly associated with the hiatal hernia. On three, phrenectomy was performed and the enlarged hiatal opening was repaired. A brief summary of these 14 cases is appended.

ABBREVIATED REPORTS OF 14 CASES OF HIATAL HERNIAE

Case 1.—Female, age 62, was examined in May, 1938.

History.—Substernal pressure, six weeks; food lodged in throat; belching of gas; pain to right of spine below costal margin, intermittent, five years. Physical examination was essentially normal. Roentgenologic examination demonstrated a small hiatal hernia. Treatment: None.

Case 2.—Female, age 58, was examined May 12, 1939.

History.—Indigestion for several years; severe anemia, two years previously; roentgenologic studies were negative at that time; severe anemia just before admission. Operation appendicitis, duodenum explored. Physical examination was essentially normal. Roentgenologic examination demonstrated a large hiatal hernia; ulceration at diaphragmatic ring. Easily reduced. Treatment: Conservative; back rest, Sippy diet, *etc.*

Case 3.—Female, age 63, was admitted December 30, 1938.

History.—Cellulitis of legs; "stomach trouble" for some time; difficulty in swallowing; burning in epigastrium; attacks of nausea and vomiting. Physical examination elicited soreness in epigastrium; otherwise normal. Roentgenologic examination demonstrated a hiatal hernia, 4 cm. in diameter. Treatment: Conservative. Symptoms persist but are not severe.

Case 4.—Female, age 38, was admitted June 5, 1939.

History.—Mild choking sensation; constriction around neck for one year; heartburn; palpitation on exertion. Physical examination was essentially normal. Roentgenologic examination demonstrated a large hiatal hernia, approximately 7 cm. in diameter. Treatment: Conservative; antispasmodic. Improved.

Case 5.—Male, age 80, was examined June 21, 1938.

History.—Abdominal pain followed by gastric hemorrhage and unconsciousness, six months previously; had been taking a nonresidue diet and alkaline powders since that time; had no complaints when examined here. Physical examination was essentially normal. Roentgenologic examination demonstrated a small hiatal hernia. No lesion of stomach or bulb. Treatment: Conservative; antispasmodic.

Case 6.—Male, age 40, was admitted March 13, 1938.

History.—Hematemesis and tarry stools, 36 hours; no stomach complaints previously; no distress. Physical examination elicited slight epigastric tenderness; secondary anemia. Roentgenologic examination demonstrated a hiatal hernia. Gastric mucosa 2 cm. above diaphragm. Treatment: Conservative; restricted diet, alkaline powders. Follow-Up, November 17, 1939: No distress whatever.

Case 7.—Female, age 58, was admitted August 16, 1939.

History.—Difficulty in swallowing for two years; liquids taken but solid food caused trouble; vomiting with above for a few weeks; nervous and had strenuous work. Physical examination was essentially normal. Roentgenologic examination demonstrated a small hiatal hernia; some pylorospasm. Treatment: Dilatation of esophagus, four times; August 22, 23, 31, and September 1, 1939. Antispasmodics. Follow-Up: Condition but little improved. Plan to section phrenic nerve.

Case 8.—Female, age 64, was admitted August 21, 1939.

History.—Severe digestive disturbance with pain and vomiting, seven months; occasionally vomited blood and had tarry stools; had lost 100 pounds in weight. Physical examination: Weight 213 pounds; mild hypertensive heart disease; postoperative ventral hernia; tenderness in epigastrium. Roentgenologic examination demonstrated a hiatal hernia; lesion on lesser curvature in thoracic portion of stomach. Esophagoscopy: Showed thoracic portion of stomach, lesion not seen. Treatment: Phrenectomy, September 6, 1939. (Ventral hernia November 1, 1939.) Good result. Follow-Up, November 22, 1939: No distress whatever.

Case 9.—Female, age 43, was first admitted in 1923.

History.—Choking; food distress; vomiting blood. Operative duodenal ulcer in 1923 (gastro-enterostomy and cholecystectomy). 1928—Disconnection of gastro-enterostomy. 1929—Symptoms returned. Conservative treatment. 1939—Belching gas; epigastric pain 15 to 20 minutes after eating. Physical examination was essentially normal. Roentgenologic examination demonstrated a large hiatal hernia. Treatment: Phrenectomy, February 14, 1939; diaphragm closed with silk. Follow-Up: Still has small hiatal hernia. No epigastric distress. November 21, 1939: Some return of symptoms. Choking sensation, not as severe as previously.

Case 10.—Female, age 57, was examined August 27, 1939.

History.—Vomiting after meals since childhood. Treated for stomach ulcer; epigastric distress. Physical examination was essentially normal. Roentgenologic examination demonstrated a hiatal hernia, 5 cm. in diameter. Treatment: Phrenectomy, September 5, 1939. Hernia closed with silk.

Case 11.—Female, age 59, was examined August 28, 1939.

History.—Nausea, pain and fulness in epigastrium for two years. Diagnosed elsewhere as malignant tumor of cardia, in 1937. Physical examination elicited tenderness throughout abdomen; achlorhydria. Roentgenologic examination demonstrated a hiatal hernia; filling defect of the cardiac end of stomach. Treatment: Phrenectomy, September 19, 1939. Hernia closed with silk. No tumor found Cholecystectomy and choledochostomy.

Case 12.—Male, age 66, was examined October 12, 1939.

History.—Stomach trouble for years; diagnosed (here) coronary, in 1933; gastric hemorrhage, duodenal ulcer in 1936 and 1937. Physical examination was essentially normal. Roentgenologic examination demonstrated a hiatal hernia. Treatment: Conservative. Nothing done.

Case 13.—Male, age 63, was examined September 29, 1939.

History.—Stomach trouble for two months (one attack four years before; roentgenograms negative); careful of amount of food—not type of food; slight upper abdominal distress; occasional nausea and vomiting. Physical examination elicited slight epigastric distress. Roentgenologic examination demonstrated a hiatal hernia. Treatment: Conservative.

Case 14.—Female, age 67, was examined in September, 1939.

History.—Cholecystectomy in 1922. Attacks of gas; occasional nausea and vomiting; had car injury two months before study—symptoms of congestive heart failure. Physical examination was essentially normal except for very mild congestive heart failure; achlorhydria. Roentgenologic examination: (1) Heart 10.5 undersize; small atelectasis; (2) multiple diverticulosis; (3) hiatal hernia. Treatment: Conservative.

The incidence of hiatal hernia is still higher than one would expect. Many more cases are being recognized each year than previously, and if the condition is kept in mind when examining the patient with atypical gallbladder and stomach complaints, a larger number of these will be discovered. Low substernal distress, inconstant and mild in character, noted especially after large meals, is an important symptom. The clinician aids in the diagnosis by becoming suspicious of the presence of hiatal hernia and requesting appropriate examinations.

It is necessary for the roentgenologist, especially, to be on the alert for this disease and to always examine the esophagus and stomach with the patient in the recumbent as well as in the upright position.

It is well to have the knowledge that the condition exists so that in the event of an increase in symptoms the patient will not undergo a needless operation for a suspected abdominal lesion or be placed upon a cardiac regimen.

Not all of the lesions are surgical conditions and conservatism can be carried out in many cases after the diagnosis is made.

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UMBILICAL AND MIDLINE VENTRAL HERNIAE *

HOWARD MAHORNER, M.D.

NEW ORLEANS, LA.

FROM THE DEPARTMENT OF SURGERY, TULANE UNIVERSITY SCHOOL OF MEDICINE; CHARITY HOSPITAL; TOURO INFIRMARY AND BAPTIST HOSPITAL, NEW ORLEANS, LA.

THE STORY of hernia parallels closely the history of surgery. In no other branch of surgery has progress been more gradual and less dramatic. Always an exterior problem, hernia has been of interest to the earliest medical authors. Its records, instead of showing how much the ancients knew, demonstrate how little essential knowledge they had concerning a problem regarded as being among the simpler procedures of modern surgery. It also demonstrates how ineffectual, and even hopeless, were the older efforts at treatment. We still have much to be desired in the treatment of herniae and the prospects for improvement in the future remain great. Undoubtedly, many more improvements, some even as important as those of Halsted[1, 2] and Bassini,[3] will be made. Advances in the surgery of hernia, which will promise more certain assurance of permanent successes, are needed. Even more often than we generally appreciate, the cure is only temporary.

Besides infection, which was one of the greatest deterrents to operation, the ancients did not know how to repair the hernial defect. Their procedures were most ineffectual. Galen[4] cut across the spermatic vessels and sac; Celsus[5] removed the testicle and sac. The description of the operation for inguinal hernia given by Paulus Aegineta,[6] who lived not later than the sixth century, states: "After placing the patient in the recumbent position and getting the skin in the groin stretched by an assistant, we make a transverse incision. Then we stretch out the incision to such a degree as to afford room for the testicle to pass through. An index finger is introduced to the back part of the scrotum to bring the testicle with the tunica vaginalis into the incision. We ascertain whether a fold of intestine be caught in the tunica vaginalis. If so, we must press it down into the belly. Then we take a large-size needle containing a double thread and pass it through the middle at the extremity of the peritoneum close to the incision. We bind the peritoneum securely. Then making an incision at the lower end of the scrotum to save a discharge, we introduce an oblong pledget and apply embrocations of oil." This was fairly characteristic of all operations described for hernia before those of Bassini and Halsted. The older operations consisted of simply tying off the sac without repairing the defect in the abdominal wall. Some ancient physicians burned the area of the external ring down through the fat layer with an actual or potential cautery, expecting the resulting scar tissue to hold back the hernia.

Petit,[7] who lived from 1674 to 1750, was one of the first to make any at-

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tempt to sew fascia over the defect. This he did by suturing the internal pillar of the external oblique to the external pillar, in an attempt to obliterate the external ring of the inguinal canal. He employed a gold suture, which may have been the origin of the term "crown-stitch." No more efficient operation was devised to repair a hernia until about 1889, when Halsted and Bassini, almost simultaneously, described their operative procedures.

Before Bassini and Halsted, even able surgeons with large practices usually regarded operation for hernia as a procedure attended with such dangers, and so little promise of success, that they recommended operation only for complicated herniae such as strangulated ones. Trusses were advocated even for irreducible herniae by Sir Astley Cooper[8] (1768–1841), a cup-shaped support being employed.

Before the sixteenth century, patients with strangulated herniae were abandoned to their fate. The physician's efforts were directed to relief of the ileus and distention, but not by release of the strangulation. No operation was performed, and measures for relief consisted in attempts at reduction by taxis and other palliative measures. During the sixteenth century, the operation of cutting the constricting ring for strangulated hernia made its appearance. According to Malgaigne,[9] Maupas, in 1551, was the first to operate for strangulated hernia. The operation consisted of a free incision into the abdomen in the region of the pubis and the reduction of a scrotal hernia. The hernia recurred. This was a great advance, but shortly thereafter Pierre Franco,[10] in 1561, described cutting the ring without incising the sac for release of strangulation. This was a further improvement because it obviated going into the peritoneal cavity and reduced, in those days, the dangers of peritonitis. However, Heister[11] (1683–1758), an outstanding German surgeon of his day, advocated attempts to reduce irreducible or strangulated hernia by taxis. If this failed, he advised clysters, even of tobacco smoke, by the use of which he said he often had surprising success. If the hernia remained unreduced for 24 hours, he advised operation, which consisted in cutting the ring. If, after opening the sac, the omentum was found to be gangrenous, it was cut off, but if intestine was found to be gangrenous, the wound was simply dressed with lint. Further advances in the treatment of strangulated hernia concerned surgery for the gangrenous intestine. Trusses continued to be the method of choice for the treatment of all herniae except strangulated ones, even to the end of the nineteenth century. Scarpa[12] (1747–1832) advised the use of a truss even after operation, because he said the hernial sac cannot be closed up to the abdomen and recurrence otherwise resulted. Macready,[13] in a monograph in 1893, still advised trusses except where operation was absolutely necessary.

The measures employed for the radical cure of umbilical hernia were just as imperfect as the older measures for inguinal hernia. Celsus[5] recommended pressure on umbilical herniae between two rulers or ligation of the entire hernia, permitting the umbilicus and sac to slough out and heal by scarring below the ligature. Paulus Aegineta ligated the sac permitting the distal part

to slough off. A method with a similar principle has been advocated even in recent times.[14]

Storer,[15] in 1867, was among the first to suture the fascia together to cure umbilical hernia. With an elliptical incision, he excised the umbilicus and sac. He worked from below upward, putting in metallic sutures as fast as he removed the sac. These sutures included all tissues of the abdominal wall. Buried sutures generally were not employed at that time. Storer operated upon a patient having very doubtful indications for surgery. Definite contraindications were present. His patient, a female, age 41, had had repeated attacks of jaundice and hepatic pain. He performed an exploratory operation through a lower midline incision. The liver was atrophic and the gallbladder distended. He closed the abdomen and the patient recovered. Shortly thereafter, the patient began to have marked distention of the umbilicus because of accumulation of ascitic fluid. He was forced to tap her more often than otherwise fearing, he said, a rupture of the umbilicus. Because of this, he chose to repair the umbilical hernia. The patient died, and on reporting the case at a meeting, he was severely criticized by a number of his contemporaries for performing a useless operation. He, too courageously, published his critics' views saying he would leave to posterity the judgment of the merits of his operation. However, present judgment cannot altogether condone the selection he made in subjecting what appears to be a hopelessly sick patient to a procedure which promised too little even for the cure of the defect for which it was intended.

Clinical Material Considered.—During the two-year period, 1937 and 1938, 112,686 patients were admitted to Charity Hospital, New Orleans. Of this number, 170 patients had herniae of the umbilicus or midline of the abdomen. Umbilical herniae accounted for 136 of these; linea alba herniae accounted for only 10, and midline incisional herniae were present in the remaining 24 patients. Fifty-seven of the patients with umbilical or midline ventral herniae were operated upon for the hernia. Forty-one of the herniae were of the primary umbilical type, 12 of the series were incisional herniae, four of which were umbilical, one epigastric, and seven lower midline herniae. Four of the cases were primary epigastric herniae.

Race and sex are important factors in the development of these herniae. The colored race seems to have a predisposition to umbilical hernia. Of the 136 cases of umbilical herniae, 97 (62 per cent) were in the colored race and 39 (28 per cent) were in the white race. During the same period, 53 per cent of the total admissions were white and 47 per cent were Negroes. Thirty of the 41 patients with primary umbilical herniae subjected to surgery were women. Twenty-two of these women were Negroes and eight of the 11 males were Negroes. The cause of higher incidence in the Negro may be explained on less meticulous care during the postnatal period, and possibly to a greater prevalence of rickets in that race. Undoubtedly, however, there is a racial predisposition to the defect in addition to the factors mentioned. The underlying causes are probably absence of the umbilical fascia in a higher percentage of Negroes than in the Caucasian race. Gorelow[16] found the umbilical fascia

deficient in 40 per cent of 300 bodies studied. So far as can be determined, no such study has been made in the colored race. This deficiency of fascia permits the development of umbilical hernia much more readily when other etiologic factors appear.

Two common operative causes of umbilical hernia are (1) obesity; and (2) pregnancy. The influence of obesity in the development of umbilical and midline ventral herniae of surgical significance, is definitely shown in this series of 41 cases. Seventeen of the patients with umbilical herniae were described as obese or very obese. Six of the 12 patients with incisional herniae were obese. Indeed, so often do umbilical herniae become serious problems in stout women, that any tendency to obesity should be considered an indication for surgery for an otherwise inconsequential hernia.

Though many of these herniae are lifelong in duration, it is surprising how many of the patients date the appearance of the hernia to a recent time. Many of these patients have small herniae, which are recognized only when another factor makes them enlarge. One-fourth of the patients gave the duration of the hernia as under two years.

Primary hernia of the linea alba is not common. There were only 10 recorded diagnoses of this condition among these 112,686 admissions. Hernia of the linea alba is usually located above the umbilicus. Primary herniae of the linea alba below the umbilicus are extremely rare. Linea alba herniae are usually small. They frequently begin by protrusion only of preperitoneal fat through a small opening; subsequently a projection of peritoneum follows as the ring of the hernia enlarges. Omentum enters the sac and may be followed by intestine. Preperitoneal fat is frequently the only hernial protrusion in epigastric hernia. In four primary epigastric herniae it was found twice. In the other instances the contents of the hernia were not mentioned. In 41 operated umbilical herniae, preperitoneal fat alone was found in the hernia in seven instances. Only one of these was strangulated. The frequency of preperitoneal fat forming the herniation in epigastric herniae is explained by the abundance of preperitoneal fat in this vicinity around the round ligament of the liver. The idea that epigastric herniae usually cause gastric symptoms by pull on omental contents is fallacious. Some further cause for the gastric symptoms must be diligently searched for before such a conclusion is arrived at. Even then, if the gastric symptoms are attributable to the hernia, the mechanism is probably reflex and not mechanical.

The ages of patients operated upon for umbilical hernia in this series varied from 3 to 68 years. The oldest patient had a strangulated hernia. Most umbilical herniae, regarded as surgical, are in patients in the middle decades of life. Thirty of 41 umbilical herniae were in the third, fourth, and fifth decades.

The type of operative procedures employed in this group of cases was varied. These 57 cases of umbilical and midline ventral herniae were repaired by 33 surgeons. The types of operative closure chosen were: The Mayo in 21; longitudinal approximation in 12; longitudinal overlapping in two; and split-

ting the rectus and separate approximation of anterior and posterior sheaths in three. In this group the umbilicus was excised in 25 instances.

In the 12 cases of incisional hernia, the records stated that a Mayo type of closure was performed; in five, longitudinal approximation of the linea alba was effected. In the remaining two cases, a longitudinal (side-to-side) overlapping closure was made. In the four linea alba repairs, the Mayo type of closure was effected in two cases, transverse approximation in one, and longitudinal overlapping (side-to-side) in one.

Infections developed postoperatively in 11 of 57 cases. In the repair of 45 of these, catgut was employed; and infections developed in nine instances (20 per cent). In one of these there was an evisceration; in one there was a very superficial infection (skin suture). In the repair of 10 cases, silk was employed—and infection developed in one (10 per cent). In two cases, the type of suture material employed was not noted. One of these developed an infection.

Seventeen of the herniae were irreducible, and 11 were strangulated—necessitating emergency operations. Two patients died following operation for strangulated herniae. The mortality, therefore, in the entire group was 3.5 per cent, and in the group of strangulated herniae, 18 per cent.

A female, age 45, was admitted with an umbilical hernia, which was said to have been irreducible for three days. It was strangulated. At operation, omentum was found in the sac. A Mayo type of closure was performed. The patient died on the second postoperative day and at necropsy a gangrenous loop of bowel was discovered.

A female, age 50, was admitted with an umbilical hernia strangulated for two days. At operation, a hernia of the Richter type was found. There was no evidence of gangrene of the bowel. The anesthetic employed was ethylene and ether. The closure was of the Mayo type. The patient died six hours after operation. Necropsy showed no definite cause of death—it being explained as a "postoperative state."

Information concerning the final results (over one year postoperative), was obtained by letter from 28 patients. Four of these admitted recurrences, and these recurrences were among 22 cases with umbilical herniae. The recurrence rate, therefore, by letter, for umbilical hernia was 18.2 per cent. Fifteen of these patients were examined, and in this group, all of whom answered by letter previously, two admitted recurrences, by letter, but examination revealed four recurrences. Of the 15 patients examined for recurrences, 11 had been operated upon for umbilical herniae. In these 11 patients, four had recurrences; thus the true rate for recurrence of umbilical hernia in examined cases was 36.36 per cent. The four patients who had recurrences were all obese. They weighed 210, 195, 170 and 286 pounds, respectively. On the other hand, two patients who did not show recurrences on examination weighed 206 and 207 pounds, respectively. It is obvious that though obesity is a potent factor in the incidence of recurrence, its presence does not always preclude a permanently good result.

Types of Repair.—In a discussion of present methods of operating for umbilical or linea alba herniae, Storer's article, 1867, deserves comment because it was, apparently, the first effort to do more than simply tie off the sac. Whether he realized the importance of getting the edges of the fascia together is not clear, since he sutured tissues *en masse*. This, however, antedated, by a number of years, the fundamental procedures advocated by Bassini and Halsted for cure of inguinal hernia. Apparently, surgeons quite generally, at the end of the last century, appreciated the necessity of closing the abdominal wall and fascia, and not merely trying off the sac. Lucas-Championniere,[17] in 1895, after realizing the frequency of recurrences, attempted to strengthen the wound in umbilical hernioplasty by overlapping the linea alba and sheath of the rectus from side-to-side. Mayo,[18] in 1899, described his operation of overlapping the fascia from above down. This has proved one of the most reliable operations for certain types of umbilical herniae, and, in follow-up studies of cases, usually proves superior to other methods by showing the minimum number of recurrences.

Since then, various types of procedures have been described, and redescribed, for the radical cure of umbilical herniae (Fig. 1 A and B). McGlannan,[19] and Stone[20] have both described operations for umbilical hernia at meetings of this Society. Other presentations before the Southern Surgical Association on ventral hernia, were given by Wathen,[21] Johnson,[22] Werder,[23] Bartlett,[24] and McGlannan.[25] The methods of hernioplasty may be included in the following types:

Longitudinal approximation of fascia.
Transverse approximation of fascia.
Longitudinal or transverse overlapping.
Relaxing incisions, arcuate above and below or laterally, to permit further mobilization of the fascia edge.
Splitting the rectus sheath and exposure of the muscle.
Mobilization of each rectus muscle, usually through a transverse incision in the anterior rectus sheath, in order to permit suturing the muscle edges under less tension in the midline. (Pfannenstiel,[26] Graser.[27])
Fascial sutures to approximate the edge of the fascia.
Fascial flaps, or sheets, to repair the defect.
Purse-string suture of the umbilical ring.

Next in importance to the anatomic plan of closure, is the material employed for closure. The controversy now current concerning the comparative value of silk and catgut probably regards one of the most important features of the surgical repair of hernia. Practically all studies dealing with this are favorable to silk, which has a lower incidence of infection and recurrence than after the employment of catgut. Undoubtedly, silk in an infected wound may delay complete healing of the wound for a longer period than if infection occurs in the wound sewed with catgut. On the other hand, silk may offer more security in that fewer infections occur when it is employed, and recurrences are less

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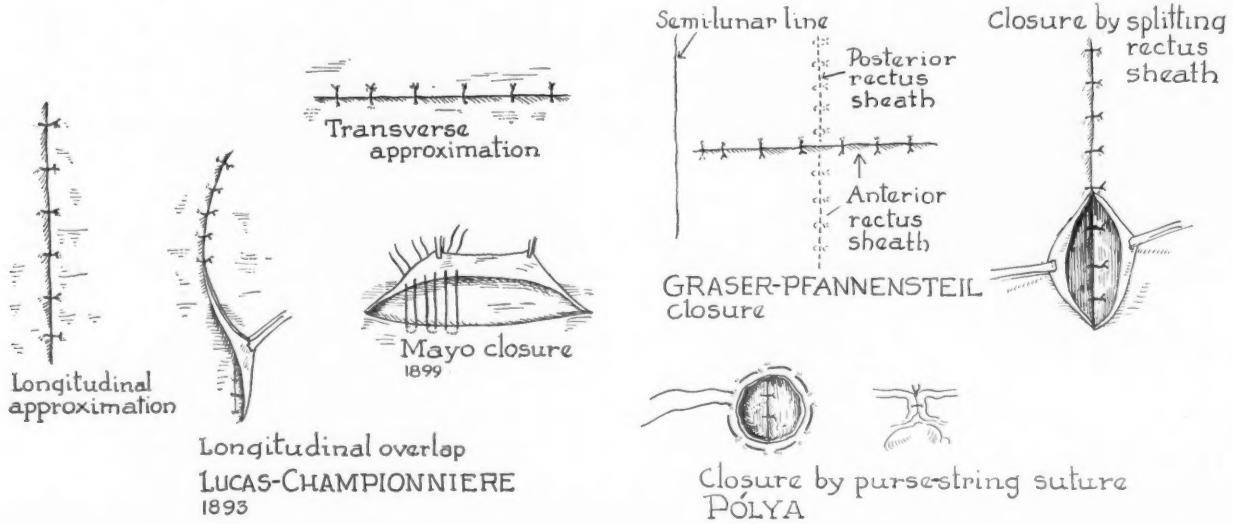


FIG. 1.—Types of closure for midline ventral and umbilical herniae. The type of operation employed depends on the size and shape of the hernial defect. Longitudinal approximation or overlapping of the fascia should seldom be employed. Transverse approximation is not as secure as the Mayo type of closure. The Graser-Pfannenstiel operation consists of mobilizing the muscles by freeing them from their sheaths and approximating them in the midline. It is an extensive procedure and is seldom indicated. Closure, by splitting the rectus sheath and separate approximation of the posterior and anterior layers, is advantageous for hernial openings long in the sagittal plane. Purse-string closure is effective for small round defects.

frequent. Meleney,[28] reporting infections occurring in a series of cases operated upon at the Presbyterian Hospital, New York, found that over a nine-year period, infections developed in 8 per cent of the wounds sewed with catgut, and in 3 per cent of wounds sewed with silk. The same author reported that, in 176 inguinal herniae sewed with catgut, the incidence of infection was 5.1 per cent, while following 303 operations for inguinal hernia in which silk was employed, the incidence of infection was 2.3 per cent.

In repairing the 41 cases of primary umbilical hernia reported herewith, catgut was employed in 32, with five infections (15.6 per cent); and silk was employed in 10 with one infection (10 per cent). Series of cases with follow-up adequate to show final results are few. Many factors are involved in recurrences, including the size of the hernia, age and weight of the patient, the incidence of infection, the suture material employed, and, not the least, accurate approximation of good tissue without undue tension at operation. Some of these factors may be favorably influenced, others are unchangeable. In estimating results it is difficult to determine the influence of one factor alone. Because of this, it is not easy to settle, unquestionably, the important question of the advantages of one suture material over another. Simmons[29] reported the results of operations for umbilical hernia performed at the Massachusetts General Hospital. There were 15 small congenital umbilical herniae, 43 large umbilical herniae, and 12 strangulated herniae. Of 10 followed cases of small umbilical herniae, none recurred. Of 39 followed cases in which large herniae, or strangulated herniae, had been repaired, the herniae recurred in 10 (22.2 per cent). Where the Mayo operation had been performed, the hernia recurred in 10 per cent. In 14 cases, where the ring was closed by longitudinal approximation, the rate of recurrence was 42.8 per cent.

Statistics which better show the influence of suture material are found in the study of Longacre,[30] and Parsons,[31] from the Presbyterian Hospital, New York. In 925 hernioplasties of all types, in 752 patients, Longacre found the incidence of wound infection in wounds repaired with silk to be 2.55 per cent, while infection occurred in 15 per cent of wounds repaired with catgut. In the entire series, silk was employed in 496 hernial repairs and catgut in 270. The incidence of recurrence of the hernia was as follows: Silk group, 3.4 per cent; catgut group, 12.5 per cent.

It was hoped that the use of autogenous fascia as a living suture, as proposed by Gallie and LeMesurier,[32] would improve the outlook for more permanent results in operations for hernia. Free fascial sheets, with edges cut into many tails, and employed as sutures, were utilized by Gallie[33] for repair of large defects. Careful postoperative studies of repairs with fascia are few. It is true that usually only the more difficult hernia operations are repaired with fascia. Surprising, if discouraging, are the results reported by Burdick, Gillespie and Higginbotham.[34] In 1,153 hernia repairs with fascia, the incidence of infection for autogenous fascia, homologous fascia and ox fascia was 7.9, 12.8 and 12.1 per cent, respectively. In 975 followed cases, of all types of hernia, there were 284, or 29.1 per cent recurrences after the use of fascia. These authors do not regard fascia as a method of choice for more

difficult hernia repairs. Since adopting the silk technic they employ fascia only in the form of a pedicle flap, as described by Wangensteen,[35] for large ventral herniae. In my experience, silk is the material of choice in the repair of herniae.

Preoperative preparation is a very essential part of the treatment of certain umbilical herniae. Large herniae, which have remained irreducible for any length of time, are said to have lost their right of domicile. There exists an unfavorable disproportion between the size of the abdominal cavity and the contents. Especially if the patient is obese and the hernia large, reduction and closure without tension may be very difficult. Even after replacement is accomplished and closure of the abdomen effected, difficulty may be encountered subsequently with dyspnea and cyanosis and sensation of oppression experienced by the patient. This phenomenon is more prone to occur after repair of upper abdominal herniae than after closure of an equally large hernia in the lower abdomen. It is due not only to an increase in intra-abdominal pressure and interference with the descent of the diaphragm, but also in upper abdominal herniae, to a restriction in the flare of the costal borders, and thus a limitation of that part of expansion afforded by the increase in intrathoracic space. Patients with large ventral herniae should be subjected to a preoperative period of preparation, lasting from a few days to a month. In this preoperative period, not only should attempts be made to improve the general condition of the patient, but specific attention should be directed to improving the circulatory system and kidney function. If there is any question concerning the heart, special studies should be made and cardiac stimulants prescribed. At the same time, efforts to reduce the weight of the patient should be instituted.

After the hernia is reduced, a binder should be placed on the patient to maintain reduction at all times. Elastic supports made of material, such as "Lastex," result in increasing intra-abdominal pressure. If the patient cannot stand pressure, at first, short periods and relatively light pressure should be used. The duration and degree of pressure is gradually increased. The object is to subject the patients to increasing abdominal pressures so that they may develop a tolerance to any additional pressure which may result from the replacement of the hernia and closure of the defect at the time of operation. Periods of preoperative preparation should last from a few days to several weeks. These essentials were, long ago, appreciated and advocated by Graser,[27] Hahn,[36] McGlannan,[19] and others. If the hernia is large and remains irreducible, Hahn has advocated performing the operation in two stages. At the first stage, the sac is dissected free and opened and after adhesions are separated, reduction is accomplished. If there is difficulty making a closure of the fascia at this time because of tension, only the skin is closed and at a subsequent date a secondary operation is performed to obliterate the hernial defect. In the interval between the two operations, the patient undergoes a period of preparation in which increasing extra-abdominal pressure by binders is employed.

A two-stage procedure allows the partial correction of factors which prevent closure without tension. In the interval between operations, the patient

can be accustomed to increased intra-abdominal pressures by binders and the relative disproportion between contents and abdominal cavity can be corrected partially by weight reduction. The recti muscles can be accustomed to their normal anatomic relation of juxtaposition by keeping the hernia reduced and keeping the muscles approximated by a binder. A two-stage procedure is more often indicated for large herniae, particularly long oval hernial openings, with the long diameter in the vertical axis of the body. Here, one would be forced to make some sort of closure in the line of the long axis, that is, a type of longitudinal approximation in the vertical plane.

Choice of the Type of Operation for an Umbilical or Midline Ventral Hernia.—The recti muscles arise from the xiphoid and from the costal car-

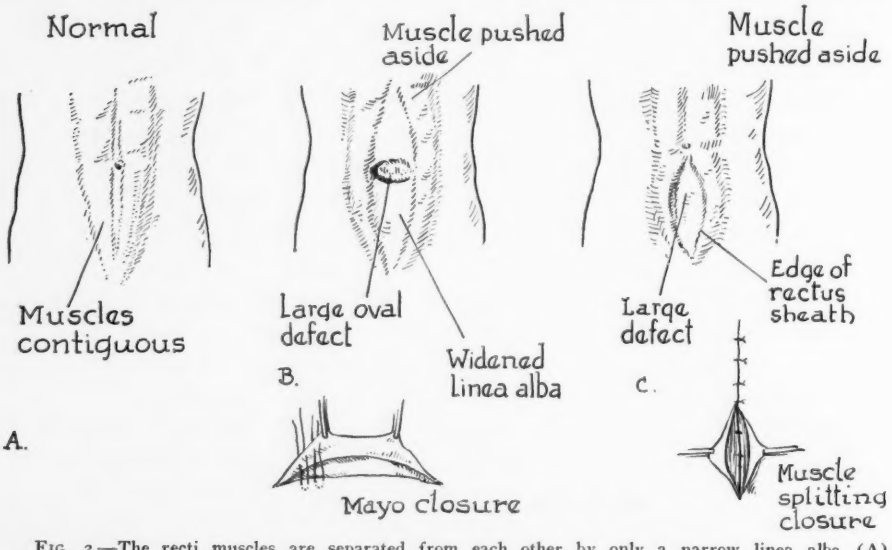


FIG. 2.—The recti muscles are separated from each other by only a narrow linea alba (A). Umbilical herniae, like herniae elsewhere in the midline, are due to defects in the linea alba. The rectus sheaths and muscles are pushed aside by enlarging herniae. It is desirable to bring the muscles back to their normal position. However, the shape of the defect, the size of the patient, and other considerations are important in choosing the type of operation for a particular hernia. In large, oval defects, wide transversely, the Mayo closure is most efficient (B). In defects large in the sagittal plane (C), the Mayo operation is not applicable, and splitting the rectus sheath with separate approximation of the posterior and anterior layers offers a strong closure.

tilages of the fifth, sixth and seventh ribs, and insert by tendons into the crest and the symphysis of the pubis.

Anatomically, the two recti muscles, within their sheaths, are close together with only a narrow intervening linea alba. The direct course of the muscles is deviated by large herniae. There are three factors mitigating against the reapproximation of the muscles at operation. One is the disproportionate size of the abdominal contents to the abdominal cavity in fat people, even when the hernia is reducible; another is the lateral pull of the lateral abdominal wall muscles; and a third factor is a broadened linea alba which itself disturbs the relationship of the border of one rectus sheath to another. In midline herniae the intact rectus sheath and rectus muscles are pushed aside by the hernia. The rectus sheath does not give way. The two sheaths are separated by the expanding hernial ring (Fig. 2). These facts are important in selecting a type of closure.

Not all midline ventral herniae can be closed most effectively, by the same type of operation. The type of operation most desirable is always the one offering the most anatomic closure with the least tension on the suture line. Many factors are involved including the size (weight) of the patient; the quality of the tissues; the location of the hernia in the upper middle or lower abdomen; and most important of all, the shape and size of the abdominal wall defect.

In repair of herniae between these muscles, the most desirable anatomic closure would be to reapproximate the sheaths of the muscles to form a new linea alba. This type of closure, however, has the disadvantage of constant counter tension on it from pull of the lateral abdominal muscles. The linea alba is a line on which the lateral abdominal muscles insert. Contrasting force is constantly being exerted at this line.

The defects of umbilical herniae are frequently almost round. Further stretching of the linea alba by obesity and enlargement of the hernia often results in distortion of the opening into an oval. When small, the defect can be closed by any number of methods, and the result can be expected to remain permanently good. If longitudinal approximation is chosen, the edges of the linea alba must be carefully freshened by cutting away the areolar tissue overlying the stronger fascia, and the opening must be converted into a biconvex lens-shape.

Since umbilical herniae develop by eccentric expansion of the defect, Pólya[37] advocates closure of certain umbilical hernial defects by exerting an exact counter force. To accomplish this, he puts in a purse-string silk suture around the hernia opening and drawing this tight, closes the opening, getting equal tension from all sides (Fig. 1). I have employed this operation for a small umbilical hernia, and found the closure easily accomplished and very effective. Several reinforcing silk sutures are placed after the purse-string is tightened and tied.

In large, obese women, with protuberance of the abdomen, the recti muscles are more widely separated than normal. A wide, but fairly strong, linea alba effectively forms the abdominal wall above and below an umbilical hernial opening in such patients (Fig. 2). Here, any type of longitudinal approximation would result in considerable encroachment on the abdominal space. Part of the linea alba would have to be sacrificed to convert the opening into a shape which would permit even approximation of the fascia. In this type, a Mayo closure offers the best promise of lasting success. Less tension results. It is lateral tension more than longitudinal which embarrasses the cardiorespiratory system. Longitudinal tension can be relieved by merely flexing the patient, and thus shortening the distance between the xiphoid and the pubic bone.

In long, oval herniae, with the long axis in the sagittal plane, transverse closure may be undesirable because it may be difficult or impossible to perform. In nine cases of midline herniae, I have effected closure by the following procedure: In one of these patients the hernia was huge, as large as a man's head, with a defect 10 cm. in width and extending from the umbilicus to the

symphysis. The technic is as follows: A longitudinal incision is made extending well above and below the hernia. If it is an umbilical hernia, the skin incision curves around one side of the umbilicus and comes back to the midline (Fig. 3). The umbilicus is not removed, but is turned aside, attached to one skin flap. It is sewed down to its normal position on closing. The incision extends down to the linea alba above and below the hernia, and the sac is carefully dissected free from surrounding tissues down to the aponeurotic ring. The subcutaneous tissue is carefully dissected away from the linea alba and the anterior sheath of the rectus. An incision is made through the midline and, after entering the abdomen, the incision is extended through the hernial ring and sac, carefully guarding against injuring any contents of

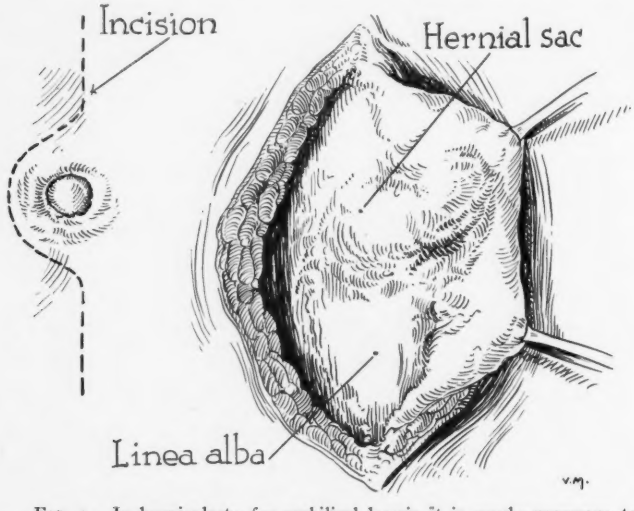
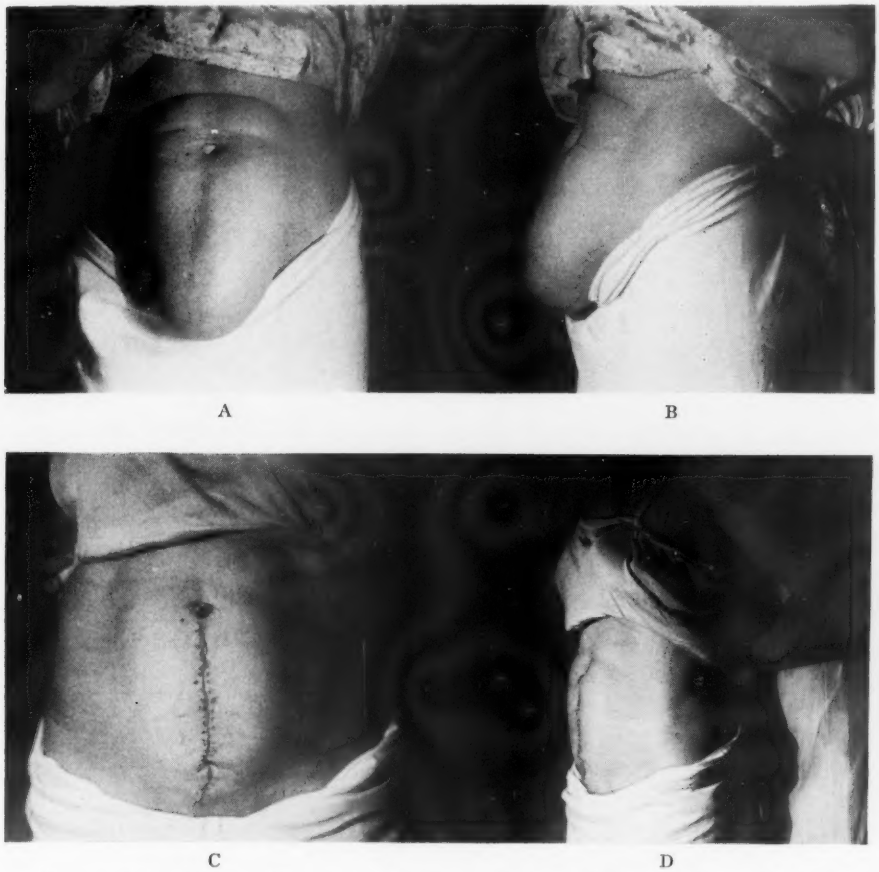


FIG. 3.—In hernioplasty for umbilical hernia it is rarely necessary to remove the umbilicus. The umbilicus may be removed to advantage in massive herniae. In smaller herniae it may be left on one skin flap and sutured in its normal position on closing of the wound.

the hernia. The hernial contents are restored to the abdomen. The sac is removed. The edges of the hernia ring are cut away so as to remove any fat and areolar tissue. The rectus sheath is incised, in order to expose the edge of the muscle. The sheath on each side is then cut longitudinally at the junction with the linea alba, and the muscle is exposed for a considerable distance even above and below the hernial opening. The posterior rectus sheath is then closed with interrupted silk sutures. The edge of the muscles may then be approximated with a few sutures, or, the edge of the muscle may be caught in the same sutures which approximate the aponeuroses. Then, the anterior rectus sheath is closed with interrupted silk sutures. It is important to go well above and below the hernial opening, in order that the closure may be uniform and the slight necessary tension not irregular.

Gersuny[38] described a similar procedure in 1893. Apparently, it is uncommonly employed. It is anatomic and has a number of advantages. In my experience with it (nine cases) it has proved of decided value in long, oval, midline herniae, with the long axis in the vertical plane. It has these ad-

vantages: First, in large, longstanding herniae, after dissecting the sac free when the rectus sheath is opened, there is no doubt that true, good fascia is exposed. The good fascia is thus not easily confused with a tough scar which might be regarded as the strong edge of the ring if a single fascial layer is intended. Second, in closing posterior and anterior layers of the muscle separately, the tension is divided onto two lines of suture instead of



A B C D

FIG. 4.—A very large midline ventral hernia. A and B, before operation; C and D, after operation. The patient, age 47, had a celiotomy, elsewhere, 18 years previously. The hernia appeared soon thereafter, and has been present since. The defect between the rectus muscle was 10 cm. wide, and extended from the umbilicus to the pubis. The patient was subjected to a period of preoperative preparation during which extra-abdominal pressure was applied. The closure was effected by splitting the rectus sheath. Special fascial bands were placed to anchor the linea semilunares together (Figs. 5, 6 and 7).

one. The anterior suture line is under slightly more tension than the posterior. Third, the muscles are brought nearer their normal position.

In the case of a very large hernia (Fig. 4), I have strengthened this type of repair with additional fascial strips transversely across the wound attached with silk to the anterior rectus fascia, far laterally, near the linea semilunaris (Figs. 5, 6 and 7). The purpose of these fascial bands is not only to strengthen the wound where they cross, but, more important, to relatively fix one semi-

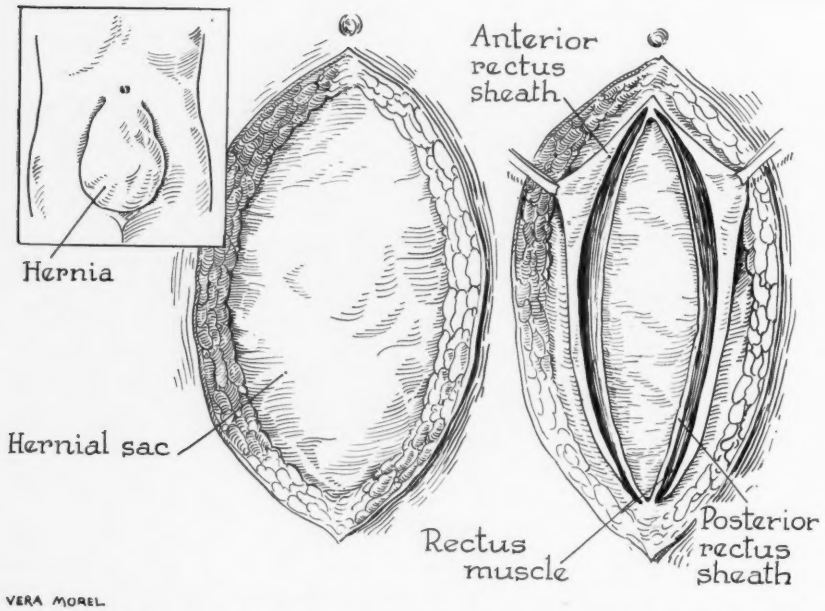


FIG. 5.—This, with Figures 6 and 7, illustrates the type of hernial repair applicable for the large ventral hernia, such as in Figure 4. After incising the skin, the subcutaneous tissue was dissected from the sac, and laterally to the rectus sheath on either side. The firm edge of the hernial ring was exposed. The rectus sheath, on each side, was split on the mesial border. The muscle was thus exposed. After the hernia had been reduced, the posterior sheath of the rectus was approximated with interrupted silk sutures in the midline (Fig. 6). Several sutures approximated the muscles. After closing the anterior sheath of the rectus, fascial strips were placed transversely, as indicated in Figure 7. These were sewed to the anterior rectus fascia near the linea semilunares. The main purpose of these strips is to anchor one semilunar line to the other, and thus relieve some of the tension on the hernial closure.

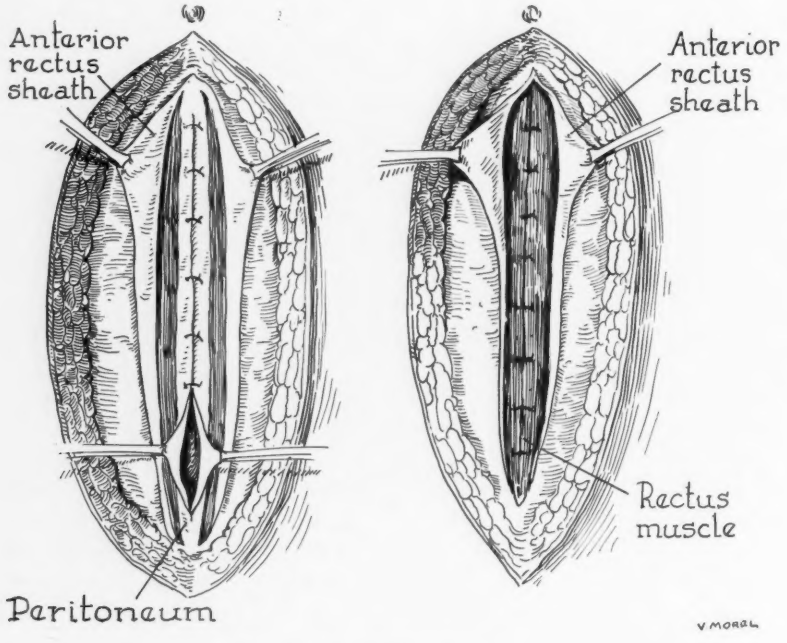


FIG. 6.—See legend under Fig. 5.

lunar line to the other, and keep them from separating, thus obviating any tendency to increased tension on the suture line by the pull of the lateral abdominal muscles. The tension, at the approximated fascia, is relieved by putting some of the pull further out near the linea semilunaris. These strips were placed by inserting them transversely across the wound. They went into the anterior rectus sheath and out, and in and across under the approximated anterior rectus sheaths and out, and in and out. The fascial strips were sewed to the anterior rectus sheath with silk at each place it went in, but at some distance lateral to the midline. After one side was sutured, the

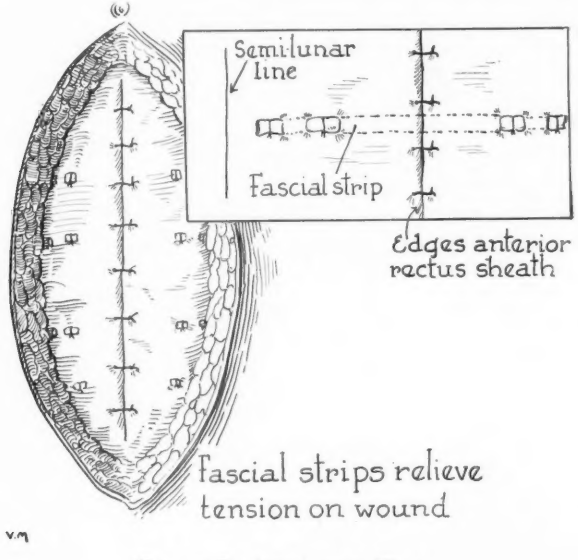


FIG. 7.—See legend under Fig. 5.

strip was put under slight tension, and the opposite ends were sutured to the anterior rectus sheath. Four strong fascial bands were so placed. In this way, the fascial strips tended to hold the lineae semilunares toward each other. This operation is not recommended for repair of herniae with oval defects, wide transversely, and with a wide linea alba, where a Mayo type of closure can be employed to better advantage. It is best applied to defects longer in the long axis of the body where herniation is pushing the muscles apart.

In order to experimentally test the use of fascia as described, it was tried on the dog. One-half the width of the rectus muscle was removed, and the closure was effected by splitting the rectus sheath of the opposite side and approximating the posterior and anterior sheaths separately. Three fascial bands were placed as described, and anchored with silk, near the semilunar line. Three weeks later, examination showed the wound well healed. The fascia bands were living and apparently effective.

Proper postoperative management after hernioplasty for umbilical or midline ventral hernia is most important. The necessity of preventing ileus or

any distention, which might increase the tension on the suture line or embarrass respiration further by increased intra-abdominal pressure, is perfectly obvious. It is essential to restrict intake by mouth and employ constant Wangensteen's suction until any danger of ileus is passed. A binder should be applied to the patient as a protection from undue strain. It is probably a good measure to continue the use of this binder for at least two to six months after the operation, depending upon the size of the hernia. Obese patients should be cautioned that the danger of recurrence is increased by gain in weight. They should reduce, if possible. The period of rest in bed after a hernioplasty is variable. A safe period of rest in bed is from two to three weeks.

SUMMARY AND CONCLUSIONS

A brief review of the major advances in the surgical treatment of herniae is given. Since anatomic closure of hernial defects was described by Bassini and Halsted, it is realized that to cure hernia, the closure must be accomplished by approximating fascia to fascia, with a minimum amount of tension. Nevertheless, the incidence of recurrence remains high and hernioplasty is still not a perfect operation.

Various technics are described for closing the defects in umbilical and midline ventral herniae. For small umbilical herniae, purse-string sutures or a number of other types of closure may be employed. For larger herniae, some type of longitudinal approximation of the edges of the rectus sheath or the linea alba is desirable. In very obese patients, with a widely spread linea alba, the Mayo operation is the least extensive and most efficient procedure. For closing hernial defects, long in the longitudinal axis of the body, with the hernia separating the recti sheaths, the Mayo operation is not applicable, and a classic repair must be made to approximate the abdominal wall in the midline. For this closure, the best procedure appears to be splitting the rectus sheath and separate closure of the posterior and anterior sheaths of the recti.

A method of relieving tension on the line of closure of the fascia is described. It consists of implanting transverse strips of fascia attached at some distance lateral to the line of closure. These prevent expansion of the relative distance between the linea semilunaris and thus tend to prevent increased tension on the suture line approximating the rectus sheaths.

The superior advantages of silk in the repair of hernia are emphasized.

It is rarely necessary to remove the umbilicus, in umbilical hernioplasty. The umbilicus can be saved on one edge of the skin. Its replacement has the advantage of being more cosmetic.

Fifty-seven cases of umbilical and midline ventral herniae operated upon at the Charity Hospital, New Orleans, were discussed. Umbilical hernia was more prevalent in the Negro race. A high incidence of obesity is found in patients requiring surgery for umbilical and midline herniae. Infections were more common after the use of catgut than after the use of silk. The Mayo

operation was most commonly employed. Next most frequent type of closure was longitudinal approximation of the fascia. Seventeen of the herniae were irreducible and 11 were strangulated. There were two deaths in the entire group and these followed operations upon strangulated herniae. Efforts to determine the end-results were not entirely satisfactory. The final results were ascertained in 11 cases following repair of an umbilical hernia. The hernia recurred in four instances.

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DISCUSSION.—DR. BRADLEY L. COLEY (New York, N. Y.): While I do not feel competent to discuss Doctor Guthrie's paper, I am very much interested in the subject, and wish to congratulate him on his presentation of it. These cases of hiatal hernia are usually treated at a general hospital but I believe if we keep a sharp lookout, we may find many more of them in our own practice.

Doctor Mahorner's paper was most interesting, and ably presented. We believe that these large ventral herniae should be given a longer period of preoperative hospitalization than was heretofore deemed necessary. While undergoing a careful preoperative study, a low residue diet should be given. In getting the patient accustomed to the supine position, I believe, we have obviated some of the previously encountered pulmonary complications. Then, we have followed the lead of Potter, in that pitressin has been employed in these cases for from 48 to 72 hours after operation.

Relative to the suture material: For more than 20 years we have used kangaroo tendon, chromic catgut, living fascial sutures, and patch transplants;

running the whole gamut of changes of technic. We feel that the greatest single step forward has been in the adoption of the *silk* technic. The following lantern slide covering the classification of 1,000 consecutive cases operated upon at the Hospital for Ruptured and Crippled, in all of which silk was used, was then demonstrated by lantern slides and gave some conception of the percentage of recurrences. The latter is based upon cases actually followed. While the statistics may not seem credible, they are accurate. In fact, it has always been our policy to lean over backwards in presenting our statistics so that there may be no room for criticism. We do not regard a letter from a patient as satisfactory evidence of his condition. Each one is recalled to the clinic and examined not only by the surgeon who performed the operation but by a group of the staff members. Therefore, I feel that you can accept my statement without reservation, that the adoption of silk has resulted in a distinct improvement in our recurrence percentage rate. We know that it has markedly reduced the incidence of postoperative wound infection. Also, we have been impressed by the lack of reaction of the tissues to silk as compared to catgut.

Dr. Isidore Cohn (New Orleans, La.): One thing I noted in both Doctor Mahorner's and Doctor Coley's statistics was the infrequency of epigastric herniae. Doctor Mahorner reported only four epigastric herniae and Doctor Coley had only seven. I believe these are a great deal more frequent than these statistics would indicate, and I think this is due definitely to the particular thing—that we do not routinely examine the abdomen for epigastric herniae. If we would palpate in the midline from the ensiform down, as some of us do, we would find a great many more.

We use practically that same method of opening the anterior sheath and using it as a second layer, as described by Doctor Mahorner. We have been doing that for seven or eight years.

Dr. Charles R. Robins (Richmond, Va.): I am much interested in the subject of ventral hernia. I will comment on only one thing, and I do this because I do not believe it has been adopted very generally. In these elderly, obese women who have those enormous herniae, any technic has to be carried out under tension.

Some four or five years ago there was published an article on the treatment of these herniae by suspending the patient in a sling which goes under the body and is attached to a line which runs over a pulley and on which hangs a weight. (Dixon, A. R.: Postoperative or Ventral Hernia—A Method for Relief of Tension After Repair. Surg., Gynec. and Obstet., **61**, 836, 1935.) It usually takes about 20 pounds to balance the muscular resistance. The advantage of this is that it brings up the loins in this way, and tends to bring the tissues together, so that when the wound is closed, instead of having to close it under tension you are able often to overlap the fascia. Where I have used it, it has worked remarkably well.

Dr. Howard Mahorner (New Orleans, La., in closing): I want to thank the members who discussed my paper. I am glad Doctor Coley could demonstrate so much better by his statistics my favorable impressions concerning the use of silk. It is my choice of suture material in these cases. So far as the type of operation described is concerned, it, incidentally, is not entirely new. Splitting the rectus fascia was first described in 1894 by Gersuny, of Vienna. Undoubtedly it has been employed many times since. Although one may have the impression he is using something different, in this instance some of the essential features have been described before.

HEMOLYTIC JAUNDICE *

ADDISON G. BRENIZER, M.D.
CHARLOTTE, N. C.

HEMOLYTIC JAUNDICE is a disease primarily due to increased fragility of the erythrocytes and characterized by anemia, increased destruction of the red blood cells, acholuric icterus, splenomegaly and a pronounced increase in the reticulocyte count. Doan[1] states positively that "true hemolytic icterus is always the manifestation of an inherited constitutional defect, characterized, when clinically active, by recurring crises of deglobulization, anemia, increased erythrocyte fragility, and unusually high reticulocytosis, acholuric icterus and splenomegaly." As Nesler[2] aptly says: "Both congenital and acquired forms of the disease are recognized. The principal difference between the two is the age at the time of onset; in general, the course of the acquired form is more severe." The diagnosis is made from the history, the detection of a symptomless mild jaundice (more or less severe) and an enlarged spleen, and from laboratory examinations. The urine does not show bile, but contains an excessive amount of urobilinogen and urobilin, as do the feces. The icterus index may be increased to 100; the indirect van den Bergh reaction is positive, the direct reaction is negative. The reticulocyte count is between 5 and 35 per cent in most cases, but may rise as high as 95 per cent. The fragility of the erythrocytes is increased to between 0.6 and 0.4 per cent (normal 0.42 to 0.3 per cent).

The following is a citation of four cases of the congenital (familial) type and two cases of the so-called acquired type. The six cases illustrate most of the findings in hemolytic jaundice (icterus, anemia).

Case 1 is the brother, brother and uncle, respectively, of Cases 2, 3 and 4. He (brother, brother and uncle) died in the hospital while being prepared for a splenectomy. The fact that he died "while being prepared" for a splenectomy might be explained in my Case 5 of the so-called acquired type and the two cases recently cited by Sharpe and Davis,[3] where hemolysis was increased by the giving of blood.

I continue to use the adjective "so-called" before the acquired type because I am taking Doan's word for it that among the cases followed by him, when properly tested, the congenital and acquired types seemed to be the same entity more or less exaggerated, differing also in the time of appearance. From the standpoint of heredity, he claims that the disease may be passed without being made evident in the individual, certainly not recognized without hematologic studies. The two acquired cases recited in my group of six cases of hemolytic jaundice would seem sporadic and individual so far as a thorough inquiry into the two family histories would show, both as to ancestry and posterity. I must

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let the reader choose for himself a familial hemolytic jaundice, becoming evident sooner or later, or two distinct types: (1) The congenital or familial; and (2) the acquired type. My conviction is that there is an acquired type much more severe than the congenital or familial type. It is true, on the other hand, that the brother, brother and uncle, who died when he was being prepared for a splenectomy, was an acquired type until jaundice and anemia proved to the brother, sister, and brother's daughter that they had the same disease. Heretofore, they had all been treated for attacks of malaria.

CASE REPORTS

Case 1.—This patient was a brother of Case 2 and brother and uncle, respectively, of Case 3 and Case 4. He was a white man, age 38, and died at the Charlotte Sanatorium, September 29, 1920, while being prepared to be operated upon. He was jaundiced and pale, and had a large mass in his left side. He had had many attacks of jaundice, pallor, fever, and pain in his left side. This last attack was his worst. There was some confusion about whether this mass was kidney or spleen. While the case was still being considered he died.

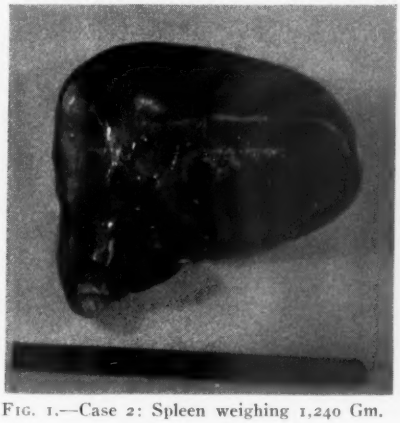

FIG. 1.—Case 2: Spleen weighing 1,240 Gm.

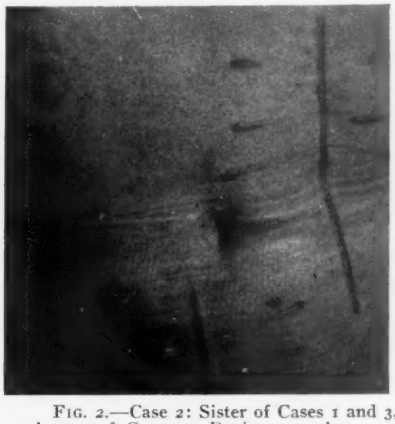

FIG. 2.—Case 2: Sister of Cases 1 and 3, and aunt of Case 4. During convalescence, developed pain in abdomen and temperature. At second operation, a widespread lymph node adenopathy was found. Refer also to Ingall's[4] case for a similar condition after treatment of dementia paralytica with malaria.

Case 2.—This patient was a sister of Case 1, sister of Case 3, and first cousin of Case 4. She was a white matron, age 27, and stated that all the above named persons were thought to have "bilious spells from malaria," and the eight-year-old niece had worms in addition. (In fact, she vomited a long, living round-worm the third postoperative day.) Married seven years, and has two children, one five and one two years, both in good health.

Patient has had "bilious spells," when she became yellow, temperature rose and she had pain across her upper abdomen. She noticed a large mass in her left upper abdomen two years ago. She says that she was toxic during both pregnancies. For four or five months, has felt exhausted all the time.

Physical Examination.—May 25, 1938: The patient was a young woman obviously anemic, her skin of a lemon-yellow color, mucous membranes, palms and soles quite pale. Spleen greatly enlarged, reaching to midline. She had already received (May 23, 1938) 300 cc. of citrated blood and the following day 525 cc. Temperature rose to 102°

and 103° F. On June 14, 1938, a splenectomy was performed (Fig. 1). She was operated upon again 22 days later (July 6, 1938), and the surgeon wrote upon his operative record: "Widespread mesenteric adenitis—probably malignant." Appendicectomy, closure. On July 20, 1938, it was noted on the chart that: "Following operation, the patient made an uneventful recovery for about ten days, and then began to have some severe abdominal pain and nausea, but obstinately constipated; had constant severe soreness and exquisite tenderness over the entire abdomen, but particularly marked in the lower right quadrant. Wound appeared clean, pelvic examination negative. These symptoms persisted with increasing intensity, and it was decided to operate. The abdomen was opened and a widespread mesenteric adenitis was found, the source being a badly infected appendix; following removal of the appendix, an uneventful recovery followed, and she was allowed to leave (July 20, 1938), much improved and wound entirely healed" (Fig. 2).

FIG. 3.—Case 3: Brother of Cases 1 and 2, and father of Case 4.

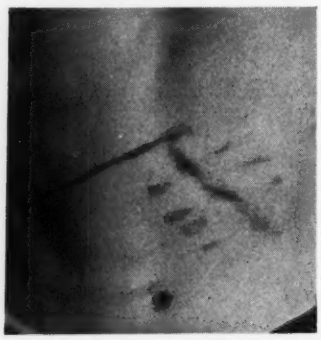

FIG. 5.—Case 3: Patient one year later, with oblique incision, well healed, but still showing, one year later, fragility of the red blood cells.

This widespread mesenteric adenitis is very interesting, showing again how, in the absence of the spleen removed 22 days before, the lymphatic system compensates or takes on some function of the spleen. A general lymphadenopathy, enlarged liver and absolute lymphocytosis during the course of paroxysms of chills and fever were reported by Ingalls[4] in a patient without a spleen, treated for dementia paralytica by the induction of malaria.

The laboratory work on Case 2 before she had blood transfusions, May 21, 1938, showed: Entirely negative urine, no bile, W.B.C. 4,150; R.B.C. 2,990,000; Hb. 11.2 Gm. per 100 cc. (73 per cent); polys. 82, lymphs. 17, large mononuclears and transverse 1 per cent; erythrocytes, great variation in size, many large and many small R.B.C., shape normal, Hb. content high.

After 825 cc. of blood, May 23, and 24, 1938: Urine, no bile, urobilinogen faintly positive. Blood: Reticulocytes 2.5 per cent; icterus index 14; van den Bergh: direct, immediate negative, delayed negative, indirect positive; no malaria parasites. Fragility test: Hemolysis begins at 0.44 (normal 0.42–0.44); hemolysis complete at 0.32 (normal 0.32–0.36).

Blood work, September 10, 1938: Hb. 85 per cent; R.B.C. 5,600,000; W.B.C. 11,700—n. 66, l. 26, e. 1, mon. 7, b. 0; platelets 275,000. Fragility test: Hemolysis begins 0.5, hemolysis complete 0.38; control 0.42–0.34.

On September 10 and December 30, 1938, patient looked quite well, and said she felt better than ever before in her life.

Case 3.—This patient was a brother of Case 1, brother of Case 2, and the father of Case 4. He was age 35 (August 25, 1938); had been married ten years, and had three

children. One child, a brother and a niece have the same trouble that he does. Large mass in left side, which is painful when he has attacks. (Fig. 3). Three attacks a year: Jaundice, pain in upper abdomen and kidney region, headache, nausea, vomiting and fever. These attacks are not more frequent lately, but are more severe. Had two ulcers of left leg that healed after splenectomy. Temperature 99° F., pulse 70, respiration 20; blood pressure 120/70; W.B.C. 8,500—n. 84, e. 2, mon. 2, s. 2; R.B.C. 3,200,000; Hb. 65 per cent; platelets 270,000. Wassermann negative; Kahn negative. No parasites in blood and stools. Urine negative; no bile. (For some unknown reason fragility test not done.) Operation, September 5, 1938: Transverse incision, splenectomy (Fig. 4).

FIG. 4.—Case 3: Spleen weighing 1,100 Gm.

Blood work, December 30, 1938: Hb. 90; R.B.C. 4,650,000; W.B.C. 10,500—n. 74, l. 14, e. 4, mon. 8, b. 0; platelets 360,000. Fragility test: Began 0.5, complete 0.4; control 0.42–0.34. Patient physically quite well, and says he feels well (Fig. 5).

Case 4.—This child, 8 years old (August 30, 1938), was the daughter of Case 3, niece of Case 1 and Case 2. She has had practically the same history of attacks as other members of the family, and everybody in the family is sure she was born with jaundice, and that her attacks are more frequent and very severe. She passed a lot of round-worms. The other two children have had no such attacks. She is now beginning to have an attack.

Blood work, July 14, 1938: Hb. 50 per cent; R.B.C. 3,100,000; W.B.C. 11,600—n. 91, l. 5, e. 3, mon. 1, b. 0; platelets 240,000. Fragility test: Hemolysis begins 0.52, complete 0.38; control 0.42–0.34.

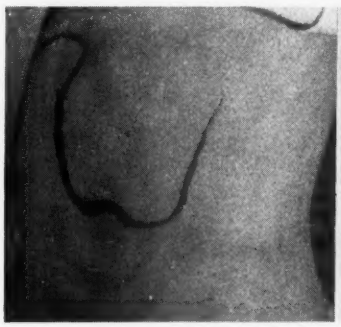

FIG. 6.—Case 4: Daughter of Case 3. Niece of Cases 1 and 2.

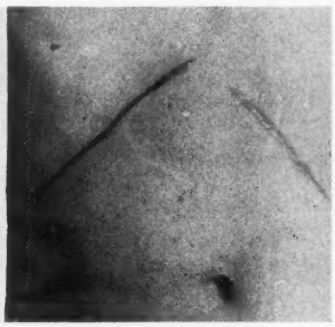

FIG. 8.—Case 4: Showing the oblique incision one year later. Still shows mild fragility of the red cells.

August 30, 1938: Patient jaundiced, pale, temperature 101° F., vomiting, large painful spleen (Fig. 6). Operation, August 31, 1938: Transfusion 300 cc. blood and splenectomy (Fig. 7). Uneventful course (Fig. 8) except a rise of temperature to 104.6° F. on the day of operation, and again to 101° F. on the third day, when she vomited a long round-worm.

Blood work, December 30, 1938: Hb. 85 per cent; R.B.C. 4,500,000; W.B.C. 17,000—n. 77, l. 21, e. 0, mon. 1, b. 1; platelets 300,000. Fragility test: Began 0.5, complete 0.38; control 0.42–0.34.

The weight of the spleen in these cases varied from 1,145 to 300 Gm. *Microscopic Examination* (Dr. L. C. Todd): (The descriptions of the three spleens were almost exactly the same). Engorgement of reticular meshes, venous spaces showing relatively little blood. There is a hyperplasia of the reticulum cells seen in areas where the hyperemia is less intense, and there is a generalized lymphoid hyperplasia of the malpighian corpuscles. Enlargement of the spleen is due to engorgement and hyperplasia. There is increased blood destruction as evidenced by erythrophagocytosis and siderosis

(Fig. 9). *Pathologic Diagnosis:* These findings are those of constitutional, hemolytic anemia ("familial hemolytic jaundice") but would have to be proven by clinical and hematologic criteria.

Case 5.—April 28, 1924: White, unmarried farmer, age 31, with marked anemia. Has been weak and pale for 18 years—the condition varies; has been very severe for past several months. Dr. William Allan: "This man presents a striking appearance—tall, thin, ghastly pale with deeply bronzed face and hands; is evidently weak, short of breath, with swollen ankles. Temperature 100° F., pulse 90; blood pressure 105/50. Abdomen full, with thin walls. The spleen extends to the crest of ilium, edge of liver palpable, abdomen otherwise normal.

FIG. 7.—Case 4: Spleen weighing 1,000 Gm.

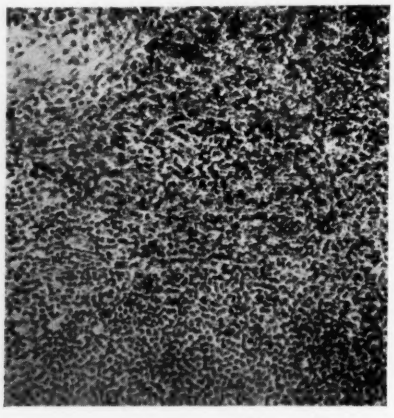

FIG. 9.—Case 4: Section typical of spleens in hemolytic jaundice. Splenic tissue filled with deglobulized red cells and pigment, increase in reticulocytes; malpighian corpuscles swollen. The spleen in hemolytic jaundice is remarkable for its lack of specific pathology.

"Blood work: W.B.C. 6,300—n. 72, l. 25, e. 1, b. 1, myelo. 1, a few normoblasts; R.B.C. 1,352,000—red cells show very marked variation in shape, size and staining; Hb. 30 per cent; color index 1.1 per cent. Fragility test: Began 0.4, complete 0.32. Blood serum tested for bile and found positive. Urine negative for sugar, albumin, indican and bile. Urobilinogen strongly positive.

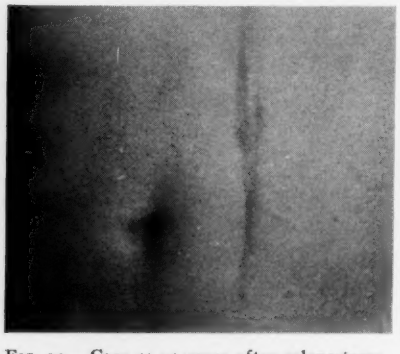

FIG. 10.—Case 5: 15 years after splenectomy. Blood picture, including fragility, normal.

FIG. 11.—Case 6: Five years after splenectomy. Blood picture normal, including fragility. The two later cases, Cases 5 and 6, show a split muscle (Bevan) incision.

"A severe hemolytic anemia, which shows a blood picture of pernicious anemia, but the clinical picture is that of hemolytic icterus."

Starting with 1,352,000 red cells and 30 per cent hemoglobin, after several massive blood transfusions—one given the day before and one on the day of operation—the red

count was 1,696,000, Hb. 30 per cent, whites 6,900. This phenomenon is further explained by Sharpe and Davis.[3] Splenectomy was performed, June 17, 1924.

Blood 14 days later: R.B.C. 4,336,000, Hb. 65 per cent; W.B.C. 12,200—n. 61, l. 29, e. 3, b. 7; no nucleated reds; color index 0.75.

January 8, 1939: R.B.C. 5,600,000; Hb. 100; WB.C. 7,900—n. 69, l. 27, e. 1, m. 3; platelets 360,000; fragility 0.46–.034. Patient in excellent health (Fig. 10).

In this case, apparently, the red cells were destroyed as rapidly as they were transfused and the patient gained nothing until his spleen was removed. The point made here is further clarified by the recent paper of Sharpe and Davis,[3] explaining severe reactions following transfusion in hemolytic jaundice.

Case 6.—August 13, 1934: A white matron, age 61. Complaint: Jaundice, paleness, weakness, gas pains in upper abdomen, large mass in left upper abdomen, which she noticed for some years. Has been getting paler, more jaundiced and weaker for the past few months. She has had her gallbladder drained; no stones were found. She has had five transfusions, receiving 2,500 cc. of blood, but continues to destroy her red blood cells. Temperature 100.2° F., pulse 100, respiration 20; blood pressure 120/70. Abdomen: Very large hard mass, emerging from left costal margin and extending below navel; notch can be felt and seems fixed under ribs (large spleen).

Blood work, August 6, 1934: R.B.C. 1,200,000; Hb. 3.9 Gm. per 100 cc. (28 per cent). Schilling hemogram: 0, 0, 2, 5, 10, 45, 37, 1; reticulocytes 40 per cent; icterus index 32.5.

There is a marked decline in megalocytes, an occasional normoblast. Reticulocytes 40 per cent, and majority are very young forms. With the rapid formation of the red cells, there must be a very rapid hemolysis of the patient's own cells to keep the red count so low. Marked hemolytic jaundice. Fragility test: 0.5–0.36. Wassermann, Kolmer, Craig, Kline and Kahn negative. Operation, August 15, 1934: Splenectomy (Fig. 11). Spleen: Weight 1,140 Gm.; 24x12x7 cm.; 180 cc. of blood drained when hilus ligation is removed. Enlargement with retention of normal shape, no nodules, no tubercles. Capsule thin and smooth, no peritoneal adhesions. Of firmer consistence than normal. Cut surface shows a deep-red pulp with widely separated and prominent follicles.

Microscopy: The follicles are widely separated; the pulp is crowded with red blood cells, though the sinuses are relatively empty. The reticulo-endothelial cells are large oval and crowded with blood pigment. The reticulum and trabeculae are not thickened.

November 22, 1935: R.B.C. 4,240,000; Hb. 11.6 per 100 cc. (84 per cent); W.B.C. 7,050—dif. (Schilling hemogram): 0, 0, 0, 5, 2, 43–49, 1.

Excerpt from patient's letter, January 4, 1939: "I have completely recovered (Fig. 11), and am getting along well."

Discussion.—Case 2: (It will be noted that this case was operated upon by another surgeon.) Shortly after her splenectomy, her temperature rose, her abdomen became tender and under the diagnosis of appendicitis, another celiotomy was performed; the surgeon vaguely described an involved appendix and mesenteric nodes due to it. Looking further, he found many mesenteric nodes which he described as cancer. Ingalls'[4] case of induced malarial infection in the treatment of dementia paralytica, where the spleen had previously been removed, not only recovered and was benefited, showing the plausibility of the treatment, but gave evidence of a general lymph node enlargement, more so on account of the absence of the spleen—the brunt of resistance to the in-

fection being taken by the reticulo-endothelial system. The eight-year-old child, Case 4, and niece of the patient, Case 2, who was operated upon at the beginning of a hemolytic crisis, likewise exhibited a rise in temperature and abdominal tenderness, which subsided during the course of her recovery. Several authors, including myself, are aware that the restoration of erythrocytes is, in these cases of hemolytic jaundice, not a gradual process, but, on the other hand, very rapid, a million or more cells gained to the blood stream while the patient is on the table during the course of the splenectomy. It would be interesting to be able to follow the changes, particularly in the bone marrow, lymph nodes and liver, under this sudden revolution brought about by the splenectomy. This autotransfusion and deluge of red cells into the blood stream is life-saving, as in Case 5 of this series, where the man was not only not benefited by transfusions, but, as was remarked at the time: "He not only hemolysed his own red cells but those from other donors."

I feel sure that further study of the entire reticulo-endothelial system, bone marrow and liver might aid us in establishing a more definite pathologic groundwork for hemolytic jaundice, especially since the organ removed, while bulky enough—plenty of meat, in other words—shows so little in its histopathology. The fact that it shows so little beyond the engorgement with remnants of erythrocytes and their pigment is one of the most characteristic points in the pathology of the spleen in hemolytic jaundice. The liver and kidney are often as much pigmented; and the liver, as indicated in the photographs of Cases 3 and 4, father and child, is considerably enlarged before splenectomy, and of normal size four months after operation. It is likewise known that gallstones and evidence of cholecystitis figure high in cases of hemolytic jaundice.

Since the removed organ, the spleen, has so little to show for itself pathologically, but since its removal usually brings about a cure of the case of the former syndrome suffered from, what, then, has stopped the destruction of red blood cells? Does their destruction, their deglobulization, depend upon their inherited or acquired quality of being more fragile? If their destructibility is dependent upon the erythrocytes' fragility, what is removed with the spleen to do away with the syndrome of symptoms formerly suffered by the patient, while the erythrocytes may still show considerable fragility and may hemolize in hypotonic salt about as they did before operation? And finally, while congenital and acquired hemolytic jaundice are chronic and a certain amount of deglobulization is going on all the time, one of the characteristics of the disease is the attacks or crises of erythrocyte destruction, when the jaundice and anemia are more intense, when the liver and spleen are much larger, in fact, when the spleen is quite large, painful and sensitive, when the temperature rises several degrees. These crises subside and those who have acquired the disease early, as in Cases 2, 3 and 4, who have had it as far back as they can remember, and the father and aunt claim the child was born jaundiced, get along fairly comfortably and live well into maturity. What, then, determines the crises in these cases? Let it not be supposed that these crises are

without danger. The reader will recall that Case 1—the brother, brother and uncle of Cases 2, 3 and 4—died while being prepared for splenectomy; that in Case 5 the patient's life was barely saved by splenectomy; and that he and Case 6 have lived 15 and 5 years, respectively, in excellent health.

The spherocyte was first described by Naegeli, and he suggested that it was pathognomonic of hemolytic jaundice. Gansslen[5] made much the same observations.

Conclusions of Castle[6]: "The following results suggest that differences in the susceptibility of various types of erythrocytes to hemolysis by hypotonic salt solution are due largely to differences in form, and not to differences in osmotic behavior.

(1) "The percentage increases in equilibrium volumes in hypotonic plasma of erythrocytes of widely different susceptibilities to hemolysis do not show significant differences.

(2) "Direct microscopic observations indicate that: (a) Hemolysis of a given type of erythrocyte is associated with the assumption of a spherical form in hypotonic plasma. (b) The more susceptible the erythrocyte to hypotonic hemolysis, the less hypotonic is the plasma necessary to cause the assumption of a spherical form.

(3) "An approximation to the percentage increase in volume necessary to cause hemolysis can be made by calculation of the percentage difference between the volume of the erythrocyte in isotonic plasma, V_o, and that of a sphere of equal surface, V_s.

(4) "In correlations of the relative degree of susceptibility to hypotonic hemolysis of a series of types of erythrocytes with their respective volume, diameter or surface in isotonic plasma, the erythrocytes of hypochromic anemia and of chronic hemolytic jaundice present exceptions.

(5) "When such correlations are made on the basis of the diameter, thickness ratio, or when absolute values are calculated according to the formula, $\frac{V_s - V_o}{V_o}$, the erythrocytes of hypochromic anemia and of chronic hemolytic jaundice do not present such exceptions.

(6) "Differences in the time necessary to cause hypotonic hemolysis of different types of erythrocytes may possibly be explained by differences in the percentage increase in volume of the discoidal form necessary to produce the spherical form in hypotonic solution."

Conclusions of Damshek, Schwartz and Gross[7]: (1) "Isohemolysins of the immune-body type were discovered in the serum of three cases of acute hemolytic anemia.

(2) "Antiguinea-pig hemolytic serum was prepared by the injection of guinea-pig red cells into rabbits. This serum possessed all the immunologic properties of the serum found in the clinical cases.

(3) "Hemolysis of the red cells of the guinea-pig *in vivo* followed the injection of this serum.

(4) "By varying the dosage of the antiguinea-pig hemolytic serum, various types of hemolytic syndromes were produced: Fulminating hemolytic anemia with hemoglobinuria, acute hemolytic anemia, and subacute hemolytic anemia.

(5) "Various types of blood pictures could be reproduced at will: Microspherocytosis; increased erythrocyte fragility; reticulocytosis; 'pseudomacrocytic' blood picture, *etc.*

(6) "The spherocyte is a small, thick, red blood cell, unaltered in volume though small in diameter, and unusually fragile to hypotonic salt solution. Our observations point to the conclusion that spherocytosis is due to the activity of hemolysin and not to an abnormal anatomic peculiarity or to a disturbed formation of cells in the bone marrow. Since increased fragility is a function of the increased thickness of the red cell, it is dependent upon the same cause.

(7) "We believe that hemolytic syndromes are due to hemolysins, possibly of different types and present in different amounts, functioning slowly in some cases and violently in others. The various blood pictures of the hemolytic anemias, namely, anemia, spherocytosis, increased fragility and reticulocytosis, are, in all probability, due to the effects of the varying activity of hemolysins, and modified by the individual's power to react.

(8) "Since the experimentally produced hemolytic syndromes and the numerous clinical types are closely comparable, the chief difference in the clinical syndromes may be a matter of the amount of functioning hemolysin present."

Surgery of the spleen has had its trials, as seen by the tables from the Mayo Clinic. Surgery now would be offered, in carrying out its greatest triumph, in the following conditions: Ruptured spleen; hemolytic jaundice, splenic anemia and hemorrhagic purpura.

The literature has again become replete with transverse incisions, instead of perpendicular ones, just as happened during the war period. The truth of the story is that we all have had an incision "bust wide open in our faces" occasionally, and then go about, as sort of an excuse, to show what we know about the abdominal wall. I am now anxious to show you some of my transverse incisions. Some of the spleens are so large that two transverse incisions run together by a connecting bar might be necessary to deliver it and restore the abdominal wall without a hernia. The reproach of a hernia is felt keenly by all surgeons, especially when secondarily repaired by another surgeon.

The trick of ligating the splenic artery, quite a large vessel, in gross splenic enlargements saves one the embarrassment of hemorrhage, if not jeopardy to the patient.

On account of severe hemorrhages from the lower esophagus in splenic anemia (Banti's disease in the late stages) before and after a splendid result from splenectomy, it has been suggested that the coronary vein be ligated, in order to lessen the venous turgescence by breaking the communication with the portal circulation.

To promote additional collateral circulation, which, in fact, is Nature's means of relieving portal obstruction, some form of omentopexy might be performed, such as that of the Thalma-Narath or the modification recommended by Pemberton.

CONCLUSIONS

(1) Hemolytic jaundice is an entity and should be easily recognized by appreciating the following syndrome: Acholuric jaundice; enlarged spleen; anemia; crises with pain and fever; increased fragility of the red cells; increase of reticular cells; urobilin but no bilirubin in the urine; normal colored stools, increased icterus index; negative direct van den Bergh with positive indirect van den Bergh reaction; and absence of itching. The congenital or familial type may embrace all cases.

(2) The blood picture: "We believe that hemolytic syndromes are due to hemolysins, possibly of different types and present in different amounts, functioning slowly in some cases and violently in others. The various blood pictures of the hemolytic anemias, namely, anemia, spherocytosis, increased fragility, reticulocytosis, are in all probability due to the effects of the varying activity of hemolysis, and modified by the individual's power to react."

(3) Splenectomy offers its greatest triumph in the treatment of (1) ruptured spleen; (2) splenic anemia (late stage Banti's disease); (3) hemolytic jaundice; and (4) purpura hemorrhagica. There are few operations as dramatic as splenectomy for hemolytic jaundice and for purpura hemorrhagica—where in the one instance there may be a gain of a million red cells, and in the other a stopping of bleeding while the patients are still on the operating table.

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DISCUSSION.—DR. J. D. RIVES (New Orleans, La.): I can make no contribution to Doctor Brenizer's discussion of hemolytic jaundice, but I would

like to ask him a question which I have found unanswerable. It seems reasonable to believe, in view of the work of Naegeli, Haden, and others, that the tendency to hemolysis in hemolytic jaundice is due to the spherocytosis. It would seem that Haden has proved that the increased fragility of the red cells is due to their spherical shape, and that only these cells are hemolyzed. It also seems well established that the spherocytosis and the accompanying increased fragility of the red blood cells persist indefinitely after splenectomy. Doctor Brenizer reports that after transfusion, hemolysis was increased and that, as a result, the liver became much increased in size. This observation has been made by numerous other observers, and we have been warned against the danger of anuria resulting from this excessive hemolysis. If the spleen is innocent and hemolyzes only the spheroidal cells, why does excessive hemolysis occur following a transfusion of blood in which there are no spheroidal cells? I have been unable to find an answer to this question, and hope that Doctor Brenizer has been able to do so.

Dr. Frank H. Lahey (Boston, Mass.): I would like to take this opportunity to mention something not particularly related to the subject, but because the question of splenectomy comes up in Banti's disease, I want to remind you of the value of heparin. While splenectomy is now performed in fewer cases of Banti's disease, heparin has been of great value to us in those cases in which the operation is indicated. Certainly, some of our troubles following splenectomy in Banti's disease have been due to thrombosis. Heparin is of value not only in cases in which the number of platelets increases from 35,000 to 500,000 in a few hours, but particularly in those in which pylephlebitis occurs during recovery. Nothing can be done for that, but here is something that offers hope. Heparin inhibits the formation of a clot, and is of definite aid, particularly, in those cases in which the splenic vein is already thrombosed and further extension of the thrombosis is taking place.

Dr. Walter D. Wise (Baltimore, Md.): Several years ago I reported, before this Association, six cases of hemolytic jaundice in the same family, with splenectomy in each instance, and I simply want to bring the record up to date for the transactions of this organization. I have now performed seven splenectomies in this family and three in another, with two other cases of hemolytic jaundice in the second family that have not yet been operated upon.

Dr. Foy Roberson (Durham, N. C.): I am very glad to hear this paper. To-morrow morning I have a little paper on "Solitary Cyst of the Spleen" and I am sorry they do not come together. I do not want to give the impression that I know anything about the spleen. However, I believe this paper should stimulate some interest in that subject. I think probably the whole question of surgery of the spleen will have to be rewritten. From 1930 to 1934, at the Mayo Clinic there were some 650 splenectomies, the majority for hemolytic jaundice, pernicious anemia and thrombopenia purpura hemorrhagica. As Doctor Brenizer has brought out, hemolytic jaundice is the one condition where splenectomy does most good. In other conditions such as in purpura hemorrhagica, where there is a tendency to remissions, splenectomy is not always indicated. I think there is another reason for renewed interest in the study of splenic surgery, namely, the effect splenectomy has on the organism as a whole. That question has especially interested me, particularly in solitary cysts, where the whole spleen has been destroyed. In solitary cyst of the spleen the whole thing is destroyed and the cysts gradually become larger and larger. What happens to that individual after splenectomy? I have

had only two cases: One was a girl, age 20, in good health ordinarily, rather robust. She began to lose weight and develop acne over the face, and the mucous membranes became pale, evidently a lack of vitamin A. After the spleen was removed the acne cleared up and she had no more digestive disturbances, yet she looked older, and the mucous membranes remained pale. What has the loss of the spleen done to this patient?

Dr. F. B. Lund (Boston, Mass.): I know nothing about the spleen, but there was a tradition in ancient times that the spleen was removed from men who were going into training for foot races and was removed on account of its weight. I do not believe that was done and can find nothing about it in ancient literature. I believe that if the ancients did remove the spleen their patients never ran again. However, they did know about the relation of the spleen to the liver. They constantly felt the enlarged spleen. One finding was described by Hippocrates. Plato, who was not a doctor but a man with a tremendously active mind, speculated about the function of the spleen, and he decided that when the liver was diseased the spleen absorbed the poisons taken from the liver and swelled, and when the liver got better the swelling in the spleen went down. It is a curious thing that although Plato probably did more to retard medicine than anyone else, in this he set forth a principle that we are still trying to explain 2,000 years later. Malaria is very prevalent in Greece now and I want to call your attention to what an American philanthropist has done for Greece. If you go to Marathon (where the fight was) you will find a small tablet in honor of the great philanthropist "Rocfelar" (John D. Rockefeller), who drained the marshes at Marathon and thus kept down malaria. There is a clinic there conducted by the Red Cross. I was there five years ago; I never saw so many spleens in my life. Three years later, I visited it again and saw hardly a spleen. There is hardly any malaria because of education and teaching the natives to fight mosquitoes and use screens. So that is what one American has done to eliminate malaria and enlarged spleens in Greece. The spleen was thought to be the organ that eliminated the black bile from the system, and the word melancholia originates from that belief. For that reason disagreeable and irritable people are said to be "spleeny."

Dr. Lucius E. Burch (Nashville, Tenn.): I merely want to call the attention of the Association to an incision brought out by our president, Doctor Singleton, in an original article, and also in an editorial in Surgery, Gynecology and Obstetrics. This incision offers an easy access to the upper abdomen. It is planned along anatomic lines, and does not interfere with important nerve supply to the part; it also has the great advantage that it is easy to close.

Dr. Addison G. Brenizer (Charlotte, N. C., in closing): Any paper I have ever presented before this Association I have presented with the idea that I would learn something from the discussion it might arouse. Of course I have not the slightest idea about why the usual elliptic red cells should become spherical before they become deglobulized. I do know merely that the red blood cells of the patient become spherical and believe that the cells from a donor also become spherical. I cannot answer that positively. I hope I can find out. I think it is likely that I shall never perform another operation, but I shall always come back here to get information and some ideas exchanged. I never give any instruction because I have little to give, but I obtain an enormous amount of knowledge from you all. Like looking at the mountains in North Carolina, if I thought it were to be my last look at them or you, I should, indeed, be overwhelmingly saddened at the thought.

FURTHER EXPERIENCES IN CONSTRUCTION OF THE VAGINA*

REPORT OF TWELVE CASES

LAWRENCE R. WHARTON, M.D.

BALTIMORE, MD.

FROM THE DEPARTMENT OF GYNECOLOGY OF THE JOHNS HOPKINS MEDICAL SCHOOL AND HOSPITAL, BALTIMORE, MD.

TWO YEARS ago, before this society, I[1] described a new and simple method of constructing the vagina. The present communication is merely a summary of the experience of those who have employed this operation.

In all, six surgeons have communicated with the author and have performed the operation, more or less according to the suggested technic. Including three cases operated upon personally, a total of 12 vaginal constructions have been performed by seven different surgeons. Follow-up examinations have been obtained in 11 of these 12 cases.

It might be well to recapitulate the technic of the operation. The principle of this operation was new, as it depended upon the proliferative capacity of the squamous epithelium of the vaginal introitus to spread and cover the walls of the newly created vaginal space. The operation attempts to reproduce the situation which exists in the fetus, in which the squamous epithelium of the external genital surface (the urogenital sinus) grows up toward the müllerian ducts and, with the müllerian ducts, thus forms the vagina. I had performed this operation first in 1928.

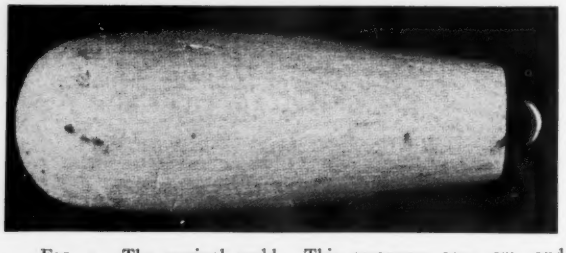

FIG. 1.—The vaginal mold. This measures 10x4 cm. and is made of balsa wood. It is well to have molds of various sizes, to fit the dissected space. The mold can be made of any rigid light material. To make it nonabsorbent, sterilizable, and prevent it from sticking to the raw surfaces, it should be covered with rubber (a condom will serve). Electroplating with silver makes an excellent mold. (ANNALS OF SURGERY, 107, No. 5, p. 844, Fig. 1, May, 1938.)

The technic of the operation is quite simple. The vaginal space is dissected, as usual, between the rectum and bladder. That concludes the operative procedure. No foreign tissue is introduced. Pinch-grafts of labial epithelium may be used but are not necessary. Into this space is fitted a smooth vaginal form made of some light, fairly rigid material, which will not adhere to the raw vaginal walls. The author has used a number of substances, but, recently, has favored a mold of appropriate size made of balsa wood and covered by a rubber condom. Such a form can be made anywhere and can be given

* Presented before the Fifty-second Annual Meeting of the Southern Surgical Association, Augusta, Ga., December 5, 6, 7, 1939.

to the patient to use when she goes home (Fig. 1). This form is placed in the vaginal space at operation and left undisturbed for three weeks, after which it is removed, cleaned and replaced. After this, the patient makes occasional visits to the doctor's office, where he can remove the form, observe the progress of epithelization and keep the wound clean. No antiseptics are applied which can injure young epithelium. The form is then worn most of the time for the next month, and after that at night for another period of three weeks, or until the vagina is completely covered by epithelium. Intercourse

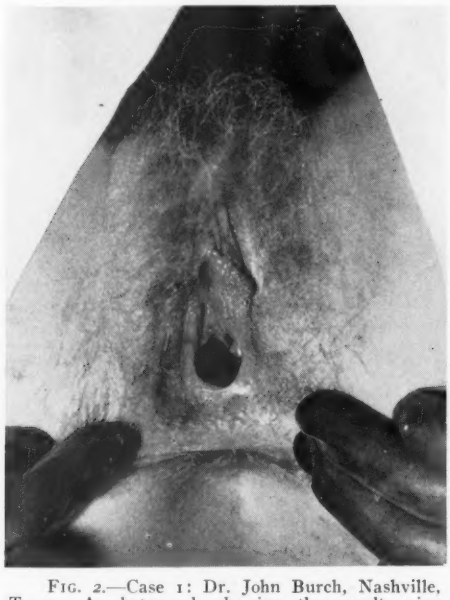

FIG. 2.—Case 1: Dr. John Burch, Nashville, Tenn. A photograph showing the result, nine months after operation. (ANNALS OF SURGERY, 107, No. 5, 852, Fig. 5, May, 1938.)

is forbidden until the space is well epithelized, which may take two to four months, at least until the mucous membrane is thick and strong.

Employing this technic, one merely dissects out the required space for the vagina, keeps it open and allows the squamous epithelium from the vaginal introitus to grow up and cover the walls of this space.

ABBREVIATED CASE REPORTS

Cases 1 and 2.—Dr. John Burch, Nashville, Tenn.: The first case was a white girl, age 18, who had cramps in the lower abdomen every month, but had never menstruated. The vagina was absent. In September, 1936, a vaginal canal was dissected and packed with loose gauze. An infection developed on the fifth day; the gauze packs were removed, and a vaginal mold covered by a rubber condom was placed. Two days later this was expelled, the space was irrigated and the mold replaced. This was then kept in place till the twenty-second day. The patient was discharged on the thirty-second day.

Follow-Up.—Nine months later: Examination showed the vagina to be large, and admitted a full size vaginal speculum; it was 8 to 10 cm. deep. The end-result is shown

in Figures 2 and 3. Figure 2 is a photograph of the vaginal orifice, nine months after the operation. Figure 3 shows the size of the vagina, as it admits a full size vaginal speculum. The result in this case was excellent. The author's technic was not followed in the beginning, as the vagina was packed with gauze instead of being kept open with a smooth vaginal form. After the gauze was removed and a suitable form used, the convalescence was normal.

Case 2.—Dr. John Burch, Nashville, Tenn.: A white girl, age 17, recently married, with absence of vagina. In July, 1939, a vagina was constructed, using author's technic. The convalescence was normal till after removal of vaginal form on the fourteenth day, when there was some bleeding. On the twenty-fifth postoperative day, there was an

FIG. 3.—Case 1: Dr. John Burch, Nashville, Tenn. The depth of the vagina nine months after operation. A full size speculum is easily introduced. The vagina is 9 cm. deep. (ANNALS OF SURGERY, 107, No. 5, 852, Fig. 6, May, 1938.)

alarming hemorrhage, about 500 cc., from an artery in the right vaginal wall. This was ligated. Transfusions were given. The later convalescence was uneventful.

Follow-Up.—Three months later, October, 1939. The vagina had healed except for some granulation tissue in the vault of the newly formed vagina. No discharge. The vagina is 12 cm. deep, and easily admits two fingers. Figure 4 is a vaginogram, three months after operation, showing the vagina distended by a condom full of barium. The constriction ring in the condom is not evident on inspecting the vagina. It probably is due to incomplete filling of the condom. Biopsy showed squamous epithelium.

Case 3.—Dr. Nathan P. Sears, Syracuse, N. Y.: A girl, age 22, engaged to be married. Diagnosis: Absence of vagina. Seven months previously, an effort had been made to construct a vagina, employing Thiersch grafts. The result was a failure. There was only a narrow tortuous sinus, 3 or 4 Mm. in diameter, where the vagina should be.

Seven months after this operation, the patient consulted Doctor Sears, who performed the first stages of a Frank-Geist operation. He made the usual "satchel-handle" flaps of skin from the thigh.

About three weeks later, the vaginal space was dissected. Because of dense adhesions and the scar caused by the first attempt to make the vagina, this dissection took two

hours and 40 minutes. One week later, the tubular graft was inserted into this space, around a vaginal mold. Two weeks later, when the vaginal mold was removed, most of the graft came with it. One week after this, the vagina was inspected, and epithelium was observed growing at the outer end. A glass form was fitted, which the patient wore continuously for several months, after which it was inserted for only an hour or so daily.

Follow-Up.—Three years later: The vagina was perfectly formed, 10 cm. deep, lined with soft, epithelial-like covering. The patient had been married one year, and enjoyed perfectly satisfactory sex relations. Biopsy and vaginogram not taken.

Final Result.—Excellent: In this case, as Doctor Sears says, although he employed my technic "more or less by accident, the result was perfect." This case brings out, in a very striking manner, the complicated steps in the other methods of making a vagina,

FIG. 4.—Case 2: Dr. John Burch, Nashville, Tenn. Vaginogram, taken by distending the vagina with a condom containing barium. The vagina is 12 cm. deep; easily admits two fingers. It is lined by squamous epithelium (biopsy). The hour-glass appearance is due to incomplete filling of the condom and is not appreciable on vaginal examination.

as contrasted with the simplicity of the author's technic. The time element is also clearly contrasted, for this young woman spent 48 days in the hospital, during which time she had three rather long and complicated operative procedures, in performing the first steps of the "satchel-handle" tubular flap operation. After she was fitted with the vaginal form, she went home in one week.

Cases 4, 5 and 6.—Dr. Buford Word, Birmingham, Ala.:

Case 4.—The unusual feature in this case was the association of a horseshoe kidney. Vaginal construction: December 9, 1937. Married: February 26, 1938. Last report: October, 1939, saying that patient is happily married and that her sexual life is "pleasant and satisfactory."

Case 5.—Colored, age 20, married. Normal female, except for absence of vagina and uterus. Vaginal construction: November 25, 1938. Discharged from hospital on fifth day. Convalescence normal. Result, excellent. Coitus normal, with orgasm. Biopsy, ten months after operation, shows vagina lined by squamous epithelium.

Case 6.—White, age 20. Complaint, amenorrhea. The patient had been receiving endocrine therapy hypodermically to make her menstruate; gynecologic examination made after a year or more of this therapy showed complete absence of vagina and uterus. Vagina constructed, May, 1939. Result, excellent. Biopsy, September, 1939, showed squamous epithelium. The vagina is well formed and commodious. As this young woman expects to be married in December, 1939, the final reports are not yet available.

Case 7.—Dr. A. A. Skemp, La Crosse, Wis.: Date of operation, August, 1938. Doctor Skemp had made two former efforts to construct a vagina in this case, but both efforts had failed. At the first operation, he had packed the vaginal space with gauze; this was followed by a small fistula which communicated with the rectum.

This patient complained not only of the gynecologic defect but also of bladder symptoms—chiefly blood in the urine. Apparently, the *urethra* had been tremendously dilated

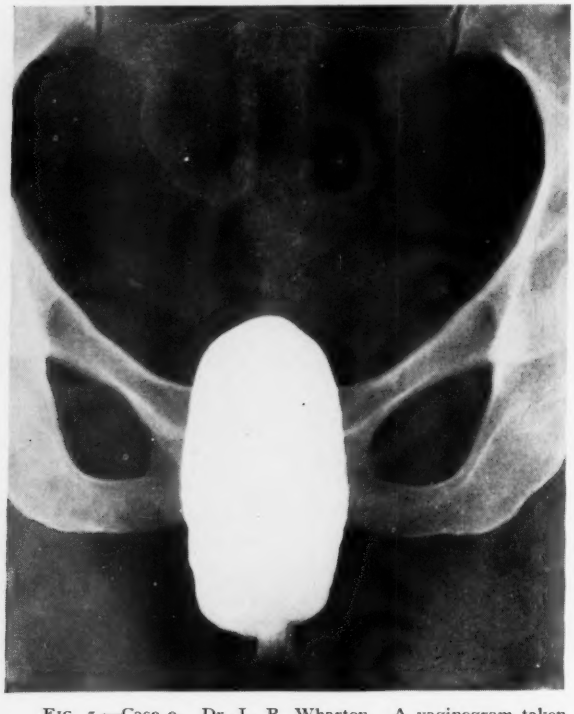

FIG. 5.—Case 9. Dr. L. R. Wharton. A vaginogram taken 1 year after construction of vagina. The vagina is large, and measures 9 cm. deep. Sexual intercourse normal; with orgasm. (ANNALS OF SURGERY, 107, No. 5, 850, Fig. 3, May, 1938.)

by coitus and it admitted two fingers. This proved to be advantageous to the surgeon, for he could orient himself during the vaginal dissection by keeping one finger in the urethra.

The last operation, performed in August, 1938, employing the author's technic, gave an excellent result.

Follow-Up.—October, 1939, 14 months postoperatively: The vagina was 11 cm. deep, distensible and painless. The patient was "completely satisfied with the results."

Cases 8, 9 and 10.—Dr. L. R. Wharton:

Case 8.—A pseudohermaphrodite, white, age 26, who wanted to be married. Operation: 1928. This case was reported in detail in the ANNALS OF SURGERY, **107,** No. 5, 842–854, May, 1938. The immediate result was perfect. The ultimate result is uncertain. In reply to our letters, the patient expresses herself as well satisfied, nine years

later. She fails to describe the situation in detail, and, since she lives 600 miles from Baltimore, a follow-up examination has not been made. Result: Unknown. This was the first patient to be operated upon, utilizing the new technic. I did not report this case earlier because I did not know the end-result.

Case 9.—A married woman, age 30. This case was reported in ANNALS OF SURGERY, **107,** No. 5, 842–854, May, 1938. The final result was excellent, except that the patient had a tiny vesicovaginal fistula, which was hardly visible and leaked only a few drops occasionally. The vagina, two years after operation, was 9 cm. deep, and was commodious (Fig. 5). The figure is a vaginogram, the constructed vagina being filled with a condom containing barium. The character of tissue lining this vagina is shown in Figure 6, this biopsy having been taken from the apex of the constructed vagina, one year after the last operation.

This case was complicated by the lack of cooperation of the patient. After the first operation, she demanded that the vaginal form be removed because she feared it would injure the rectum; this nullified, to an extent, the first operation. One year later, the second operation was performed. In spite of our warnings, she promptly had sexual intercourse immediately after leaving the hospital, three weeks after the operation. This stripped off some of the young epithelium and left her with a vagina about 7 cm. deep. One year later, the final operation was performed, securing a vagina which was 9 cm. deep (Fig. 5). At this last operation, however, the dense scar tissue made the dissection very difficult, and a small hole was made in the bladder. This was closed, a retention catheter placed and the convalescence was normal. There is still a tiny vesico-vaginal fistula, which is so small that it does not drain enough to make the patient wish to have it closed. Result of vaginal construction: Good.

FIG. 6.—Case 9. Dr. L. R. Wharton. Biopsy of vaginal wall, one year after construction; shows a lining of normal vaginal epithelium (Union Memorial Hospital, Path. No. 10,276). (ANNALS OF SURGERY, 107, No. 5, 851, Fig. 4, May, 1938.)

Case 10.—A young woman, age 21, who desired to be married. The only abnormality was the absence of the vagina and uterus. This young woman had had two former operative attempts to form a vagina, each time the vagina being packed with gauze. As soon as the gauze was removed, the cavity collapsed.

Examination showed a shallow vagina about 3 cm. deep. Operation: May, 1939. The patient was kept in the hospital three weeks, and then allowed to go to her home in Alabama, to have the necessary postoperative care under the supervision of her family physician. She apparently developed an irritation of the vaginal orifice, and the insertion of the form became very painful. She consequently neglected to wear the form, and the cavity collapsed, adhered, and finally became only 5 cm. deep. When she left the hospital, it was 13 cm. deep, and unusually commodious. Her physician had advised her to return to Baltimore as soon as she found it difficult to insert the form. She refused to do this, however, and gradually lost the benefit of the operation.

In October, 1939, she returned, requesting that the vaginal operation be repeated. The difficulty and risk of the operation was explained, as this would be the fourth dissection of the rectovesical septum. Nevertheless, she demanded that the effort be made. At operation, however, there was no plane of cleavage; dense scar tissue bound the mucous membrane of the bladder to the rectal wall. After the space had been dissected to a depth of 7 cm., a hole was accidentally made in the bladder. This was immediately closed and the bladder drained by a urethral retention catheter. Further attempt to

enlarge the vagina was abandoned. The final result was that this girl now has a vagina about 5 or 6 cm. deep, about half as deep as it was after the author's first operation. In this case, the benefit of the operation had been largely lost by the failure of the patient in neglecting the proper postoperative care. Also, this case again emphasized the extreme difficulty that attends third or fourth attempts to dissect out the space for the vagina.

Case 11.—Dr. J. Mason Hundley, Jr., Baltimore, Md.: This case was published in the ANNALS OF SURGERY, **107**, 842–854, May, 1938.

The patient was a pseudohermaphrodite, white, age 26. Testes were the only gonads; the external genitalia were of the female type; the testes were in the groins. There was no vagina. Since the patient had been raised as a girl, she wished to have a vagina so that she might marry.

Vaginal construction and an exploratory celiotomy were performed, November 10, 1937. Six days before this operation, the operator had partly dissected the space between the rectum and bladder, but had done nothing else. It happened that the author read his preliminary report of his method of constructing the vagina at a medical meeting and Doctor Hundley utilized the technic on this patient. The immediate result of the operation was successful. The patient discontinued the use of the balsa wood forms, however, and the space contracted. She, therefore, consulted a surgeon elsewhere, who stretched the vagina and covered the raw surface with Thiersch grafts. The result was apparently excellent. The patient is now married. We consider the first operation a failure, due to the failure to follow the proper postoperative care.

Case 12.—Dr. Charles Stevenson, Baltimore, Md.: This patient was a colored girl, about 15 years old, whose only complaint was the inability to have intercourse. The only demonstrable physical defect was the absence of a vagina and uterus. Operation, May, 1939: The surgeon reported that the operation was easy and completed without difficulty. The hospital convalescence was also satisfactory. There was some difficulty in the late postoperative care, however, and the vaginal forms were not used. The vagina was apparently about 4 cm. deep when the last examination was made. This operation is therefore classed as a failure.

Genito-Urinary Malformations Associated with Absence of Vagina.—This phase of the problem is not within the realm of this paper; therefore, it will be dismissed by merely summarizing the findings. In a forthcoming study, this interesting question, and the findings in these and other cases, will be presented in detail.

In these 12 cases, ten were normal females, as far as sexual differentiation was concerned. They presented no trace of hermaphroditism. Two were pseudohermaphrodites, of the male sex, with female secondary sexual features and female external genitalia. These two pseudohermaphrodites had testes, and no ovaries. They had no visible müllerian duct.

In one of the 12 cases, there was a horseshoe kidney. In a future study, we shall present a case that had complete absence of one kidney with incomplete descent of a functionless, cystic ovary; another case showing absence of one ovary; and a third, with myomata, as large as a grapefruit, developing from a uterus no larger than a thimble.

In general, every case of absence of the vagina should have a complete genito-urinary examination because of the possible association of other malformations.

Discussion.—Final Results: The author has assembled a group of 12 operations for constructing the vagina according to the suggested technic. These

operations were performed by seven operators. The final results of the operation were perfect in eight cases, unknown in one case, and complete or partial failures in three cases. In the successful cases, the constructed vaginas were lined by squamous epithelium in every case in which a late biopsy was taken. The vagina was usually 9 to 13 cm. deep, commodious and insensitive. Some of the patients expressed themselves as being completely satisfied; sexual life was normal; orgasm was definitely experienced by some of these women. In one instance, the author observed that the vagina increased in depth by 1 cm. during the first year after operation, showing that the vagina is pliable.

Operative Difficulties.—The rectovesical septum is normally soft and presents a clean plane of cleavage. In the absence of scar tissue, the rectum and vagina can often be separated by blunt dissection in a very few minutes. This step is common to all operations for constructing the vagina.

If this septum has been repeatedly mutilated, infected and scarred-over by previous dissections, it is gradually replaced by dense fibrous tissue, which binds the very delicate bladder mucous membrane and muscle to the thicker rectal wall. Hence, if one is unfortunate enough to have to try to make a vagina after two or three others have failed, he is sure to experience great difficulty in dissecting the space between the rectum and bladder. The chances are even that he will injure the bladder; in one case, a surgeon injured the rectum. One can orient himself by keeping a sound in the bladder and an assistant's finger in the rectum. In spite of this, however, the bladder will be unavoidably perforated occasionally. This emphasizes the importance of refraining from ill-advised attempts to make a vagina; a failure will only lessen the chance of future success and greatly increase the probability of vesicovaginal or rectovaginal fistula.

The Type of Vaginal Form.—In these operations, various substances have been used to construct a vaginal mold or form, such as large rubber tubing covered with layers of gauze, tongue depressor wrapped with gauze, melted paraffin poured into a condom, balsa wood molds, and molds made of the obturator of a proctoscope, plated with silver. All of these substances, with the exception of the silver form, have been covered with thin rubber (condom) to keep them from sticking to the raw vaginal walls. All of these forms have been satisfactory. It seems that the only essential features are that the form should be sufficiently rigid to resist the constant pressure of the perineal muscles; smooth, so that it will not adhere to the sides of the vagina; clean and sterilizable; nonabsorbent, so that it will allow good drainage; and light, so that it will not press on the rectum. The silver form made by Doctor Word is probably the most satisfactory, but cannot be made by everyone, and cannot be given to the patient. The simpler forms have, however, proven just as satisfactory, although we would prefer the silver form when available.

Use of Mucous Membrane Grafts.—These have not been necessary, as shown by the successes obtained when they were not employed. The author used them in his last case, but did not notice any increase in the rate of epithelization. It is possible that they may act as foreign bodies and predispose

toward infection if they do not "take." We see no objection against placing a few pinch-grafts over the end of the vaginal form, cutting these grafts from the inner surface of the labia minora. But this step is not necessary; if the vaginal space is cared for properly and kept open, the result will be good in either case.

Necessity of Retention Catheter.—We have used a retention catheter only when the bladder has been injured. An indwelling catheter has been used by about half of the operators. If it is not used, it may be necessary to catheterize the patient once or twice, but after that urination is usually normal. In one instance, the retention catheter increased the pressure on the vaginal mold sufficiently to pinch the urethral meatus and cause a slough in the urethral wall just under the pubis, at the external orifice. This did no functional damage; it is however, unnecessary.

Length of Hospitalization.—Some operators have allowed their patients to go home in five days, others eight or nine days, still others after three weeks. The results have been equally good in all cases.

Postoperative Care.—Most operators have not removed the form till the twenty-first or twenty-eighth day. It is easily removed and replaced, without anesthesia. The vaginal walls can then be examined most readily in the knee-chest position and the process of epithelization watched.

The after-treatment consists in keeping this space open and clean. One may irrigate it occasionally, using salt solution or boric acid. Weak antiseptics such as mercurochrome (2 per cent) can be applied. The lubricated form must be worn most of the time for two months. As the walls become more rigid and covered with the blue layer of thin epithelium, the form can be left out part of the time. Eventually, it can be left out permanently, after the epithelium has become thickened.

Intercourse is prohibited till the epithelium is thick and strong. This will probably take three months, although in one case (Word) the patient married ten weeks after the operation and has had normal sexual relations ever since.

Bleeding.—Hemorrhage was a serious complication in one of these 12 cases. It is imperative that the vaginal walls be completely dry before the form is introduced. The points from which one may expect serious bleeding are on the lateral walls, in the position of the broad ligaments. These areas are vascular and may require transfixion sutures. If the walls of the space are not dry, the seepage of blood may force the form out; in one case, the postoperative hemorrhage was enough to require transfusion.

Infection.—This has not been a factor in any case in which the author's technic was employed. Careful technic in the operating room, hemostasis and lack of trauma help to avoid infection.

THE CAUSES OF FAILURE AND COMPLICATIONS

(1) *Failure to wear the vaginal form:* It is essential that the space be kept open. All of our failures are definitely traced to the patient's neglect in wearing the form.

(2) *Injury to bladder or rectum:* The surgeon who is the first to separate the rectum from the bladder has an easy task; he can do the operation by blunt dissection in a few minutes. The surgeon who performs the second dissection a year or so later has a definitely harder task, as there is a good deal of scar tissue. If he fails to make a satisfactory vagina, the next operator runs an even chance of injuring the bladder or rectum. The fourth trial is almost sure to be futile.

If the bladder is injured, it is immediately closed over, using whatever tissue is available. If there is any solid vaginal mucosa, it is a great asset. One cannot very well enlarge the vagina after the bladder has been torn. Also, it may be risky to use a vaginal form, although in one case the author did place a form and got an excellent result. The patient still has a small fistula, however, just enough to leak a few drops intermittently. Injury to the bladder or rectum immediately lessens the chance of making a satisfactory vagina.

It is to be remembered that the dissection of the vaginal space is a necessary step in any operative method of making a vagina, and that the above statements would, therefore, apply equally to any surgical method of constructing the vagina. The only method of constructing a vagina which does not involve any surgical dissection is the gradual dilatation of the vaginal space by manual pressure, using a glass or metal dilator. This is probably one of the oldest procedures, having been described and practiced about 100 years ago; it has recently been reintroduced.

CONCLUSIONS

The author's method of constructing the vagina has been employed by six other surgeons. In all, 12 cases have been operated upon.

The final results have been perfect in eight cases, partial or complete failure in three, and unknown in one.

The depth of the vagina, as determined by follow-up examinations, several months to three years after the operation, has varied from 9 to 13 cm. in the eight successful cases. It has been commodious, insensitive and distensible.

The vagina has been lined by squamous epithelium. It is our opinion that this grows up from the epithelium at the vaginal orifice.

The length of hospitalization varies from five days to four weeks.

Postoperative hemorrhage was encountered in one case.

Sexual relations have been described as satisfactory to both husband and wife.

Operative injury to the bladder or rectum occurs occasionally; in 12 cases, the rectum was injured once, the bladder twice. In patients who have been operated upon several times, the rectovesical septum is a mass of scar tissue and injury to these hollow viscera may be unavoidable. One should hesitate before attempting to operate upon a person for the third time and explain the situation to the patient; at a fourth operation, the chance of injuring the bladder is very great, and the likelihood of forming a satisfactory vagina equally slight. Gradual dilatation of a small, shallow vagina, 5 or 6 cm.

deep, may make it considerably deeper, provided there is not too much scar tissue.

If the vaginal space can be dissected satisfactorily, one can almost guarantee a good result if the vaginal cavity is kept open and clear. This emphasizes the importance of operating only upon women who will cooperate in carrying out the proper postoperative treatment.

In conclusion, the above record shows that six surgeons, who had never seen the operation performed, carried out the technic and obtained good results. In 12 cases, there were eight successes, and three partial or complete failures. There were no deaths.

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CONGENITAL ARTERIOVENOUS ANGIOMA OF THE ARM*

METASTASES ELEVEN YEARS AFTER AMPUTATION

RUDOLPH MATAS, M.D.

NEW ORLEANS, LA.

THIS is the history of a patient with a congenital arteriovenous angioma (cavernous type) of the right upper extremity, complicated by grave cardiovascular disturbances; regional recurrence and seeming metastases in the corresponding right axillary scar and chest wall, *eleven* years after disarticulation of the arm at the shoulder; excision of the chief metastatic growth in the axillary scar and chest wall, with recovery and seeming freedom from recurrence or metastasis almost four years later.†

The patient evidenced an arteriovenous angioma of the cavernous type which, appearing externally at birth as a small pulsating swelling in the little finger of the right hand of a congenitally hypertrophied arm, grew upward until, in the course of 21 years, it reached the armpit. Its advance was first checked temporarily, when the patient was 16 years old, by an amputation of the forearm, after the ligation of the radial and ulnar arteries had failed to control it. Recurrence soon followed in the stump, compelling the amputation of the arm at the shoulder at the age of 21.

The case owes its chief interest to the fact that *nearly 11 years* after the amputation at the shoulder, and after the apparently complete recovery of the patient, the disease recurred as a regional metastasis in the axilla and scar of the amputation and as a separate angioma at a distance, in the corresponding side of the chest wall. The clinical characteristics and the microscopic examination of the new growths fully confirmed their histologic and clinical identity with the primary angioma of the arm amputated 11 years before. This tumor, therefore, enters that very rare group of the *metastasizing hemangiomata* which are at present claiming much of the attention of pathologists and clinicians in an endeavor to account for the seeming paradox of a histologically benign and clinically malignant tumor. In other words, a histopathologic illustration of the old fable of the "wolf disguised in sheep's clothes."

Another distinctive feature of this growth, and quite apart from its purely oncologic or tumoral aspect, is the effect that the growth, as an arteriovenous cavernoma, has had on the heart and circulation in the course of its long developmental history. When we realize what a single arteriovenous fistula

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† A preliminary report of this case in abstract was read at the meeting of the Southern Surgical Association at Biloxi, Miss. (December 16, 1936), but delayed for publication to permit of further observation in regard to recurrence and metastasis. An interval of nearly four years has elapsed since the performance of the last operation (April 8, 1936) and at the present date (January 20, 1940), the patient is in good health and, apparently, without signs of recurrence or metastases.

between a large artery and vein can do to injure and cripple the heart, we can understand how a monstrous tumor like this, which is structurally but a coarse sieve through which the arterial blood of the whole extremity is poured directly into open venous channels without the interposition of the capillary bed—must strain the heart by the enormous venous overflow that is shunted into it through the open arteriovenous channels in the tumor. The remarkable recovery of the heart and circulation after the amputation of the arm, despite the many years that the patient had been disabled by his abnormal circulation, and the new therapeutic and prognostic problems set up by the unexpected reappearance of the growth, in connection with the intrinsic interest that attaches to this rare and formidable type of vascular growth, would seem to justify the detail with which the history is herewith presented.

Case Report.—When the patient, C. C., male, age 21, first came under my care, February 20, 1925, he appeared as a rather undersized but well-proportioned youth of Israelite-American descent, five feet four inches in height, weighing about 130 pounds. He was an alert student, who had advanced at college despite the handicap of a disabled right arm and long suffering caused by the angiomatous disease. His family antecedents were excellent. There was, apparently, no sign of hereditary disease or deformity in his immediate and remote ancestry and collaterals; the father was living, normal, and without complaint, at age 58, and his mother likewise well, at age 47. He was the youngest of three children—a brother and sister, both healthy, vigorous and without complaint.

His mother stated that at birth he was, in every respect, a perfectly normal baby except for a notably enlarged right arm, including the hand and forearm, both in length and circumference, and although differing from the opposite arm in size, it was in no way large enough to attract attention as a deformity. The enlargement suggested a simple hypertrophy without deformity or edema. The enlargement seemed to involve all the parts symmetrically, even the bones feeling larger and thicker than on the other side. There was no difference in the use of the arm. Apart from this uniform enlargement, the only feature to attract attention was a small red birthmark (nevus) on the little finger of the right hand.

Although physicians were consulted, nothing was done therapeutically as there was little or no functional disability, though the limb continued to enlarge very gradually with the growth of the child. Otherwise, the baby grew and developed as any other healthy, normal child, giving no cause for anxiety until he was three years old, when he was seized with an acute metapneumonic empyema from which he had a narrow escape only after the resection of a rib and long pleural drainage.

This operation and a tonsillectomy, at age six, constituted the chief complications that were added to the patient's slowly enlarging arm, until he was 11 years old, when he began to suffer irregularly from periodic attacks of pain with extreme sensibility of the hand, wrist and fingers, accompanied by some fever. These episodes were so severe that they kept him in bed while they lasted, usually for several days, and a notable increase in the size of the arm followed each attack. These attacks were renewed at long intervals until he was 14 years old when he was prostrated by an extraordinarily violent illness limited to the affected arm. It was diagnosed acute articular rheumatism, which kept him in bed for over seven weeks. This attack involved all the joints of the fingers, hand and wrist and, judging by the description, had all the characteristics of an acute arthrosynovitis with fever and an exquisite pain on touch and motion that caused unutterable suffering. When the attack finally subsided, the hand was left permanently swollen and disabled, with all the joints of the hand and wrist thickened, stiff and disfigured by hard edematous swelling.

The true nature of these attacks is not known nor their relation to the angiomatous disease which, though unrecognized at the time, had undoubtedly existed in the hand since birth; but the periodicity of these attacks, the unilateral, regional localization, with fever and progressive swelling after each attack, are suggestive of the periodicity of elephantiasic fever and permanent lymphedema caused by Unna's streptococcus of elephantiasis nostras.

The permanent swelling, stiffness and pain in the hand, which completely disabled the arm, led the parents to take the patient to the Mayo Clinic. This was in 1919, when he was 16 years old, and where the first serious study of his case was undertaken by Dr. J. deJ. Pemberton, who recognized the angiomatous disease which had been at the bottom of the boy's pathology since birth.

I am very much indebted to Doctor Pemberton[1] for directing my attention to a summary of this patient's case while at the Mayo Clinic, in a very valuable paper on "Congenital Arteriovenous Communications," prepared by himself in conjunction with Dr. James H. Saint, which was published three years after I had operated upon the patient in New Orleans. The observations are very valuable, as they provide the essential data regarding the condition of the patient and his tumor at a most important period of

FIG. 1.—C. C., age 15: Congenital cavernous angioma (arteriovenous) of hand, wrist and forearm. Reproduced by courtesy of Dr. J. deJ. Pemberton, from photograph taken at Mayo Clinic in 1919. (Pemberton and Saint: Surg., Gynec. and Obstet., **46**, 472, April, 1928.)

his history; besides, they afford an excellent photograph of the hand and forearm before the progress of the disease compelled their amputation (Fig. 1).

Quoting the description given by Doctor Pemberton,[1] in his paper of 1928, we learn that: "The patient, C. C., was first admitted to the Mayo Clinic in 1919, when the right arm was seen to be of greater girth than the left, also exceeding it in length by 1.25 cm. A pronounced arteriovenous bruit was heard in the palm of the hand and in the right clavicular region. Roentgenograms of the hand showed what seemed to be a destructive arthritis of the proximal ends of the metacarpal bones and bones of the carpus. The radial and ulnar arteries were ligated at different intervals and divided (Doctors Henderson and Myerding). At these operations all the vessels in the neighborhood of the wound seemed to be enlarged. Following these ligations the size of the hand was reduced one-third, and the bruit and pulsations were considerably diminished." It was now evident, also, that the great bulk of the swelling in the hand and fingers was caused by the steadily growing angioma.

The patient again visited the Clinic in 1920. Six months previously, he had noticed a dark scab over the joint of his little finger, at the seat of the primary birthmark or nevus. One month prior to his arrival at the Clinic the scab broke down and a very painful, progressive ulceration followed. It was then decided that an amputation through the middle of the forearm was necessary and this was performed (Doctor Henderson) in September, 1920, on the eve of the patient's seventeenth birthday. The pathologist's report was "progressive gangrene of the fingers and half of the hand with the beginning of ulceration on the dorsal surface of the little finger." (The precise cause of the gangrene is not stated.) The amputation gave the patient great relief and made him free to enjoy outdoor sports, especially one-arm tennis, which his sensitive and long-suffering right hand had previously denied him.

Three years after this event (1923) the patient (then a student at Washington University), while going through a periodic physical examination, was told by the medical examiner that he had an

aneurysm in the subclavian region which was seriously affecting his heart. He was then advised to return to the Mayo Clinic, where he was readmitted in July or August, 1924. There he was examined by Doctors Pemberton and Henderson, and the late Doctor Brown, whose attention was chiefly directed to the right supraclavicular region, where all the signs pointed to a congenital arteriovenous aneurysm of the subclavian vessels.

Again, quoting the observations of Doctors Pemberton and Saint:[1] "A systolic pulsation was visible in the supra- and infraclavicular areas and a subdued thrill which was best obtainable in the right supraclavicular space. The stump of the right forearm, which had been amputated three years previously, was about three times larger than the normal side, tapering off toward the shoulder. The *whole arm* pulsated with each systole. A loud, continued bruit, with systolic exacerbations, could be heard throughout the right arm and up to the clavicular region, but it was most marked in the supraclavicular space. The superficial veins of the right arm were dilated to three or four times their normal size. The heart was markedly enlarged, the apex beat being visible in the sixth left interspace. The systolic blood pressure in the normal left arm was 117/56, pulse 72." In a recent (January 5, 1937) personal letter, Doctor Pemberton states that one interesting feature of the examination which was not included in the case report of 1928 was "a marked difference in the blood pressure of the right and left arms; that in the right arm being 130/85, while that in the left was 106/56." Continuing with the notes recorded in 1924: "The surface temperature of the right arm was 96.4° F., while that of the left was 91° F. The blood in the veins of the right arm was examined for differential oxygen content by the late Dr. Brown, who found the venous blood flowing from the arm to be almost arterial in character, as determined by the oxygen saturation (90 per cent for the right, 30 per cent for the left arm). Dr. Brown also tried to visualize the arteriovenous fistula, which was supposed to exist between the subclavian vessels, with a sodium iodide radiopaque solution, but this failed to exhibit any arteriovenous communication, presumably because of the unusually free collateral circulation."

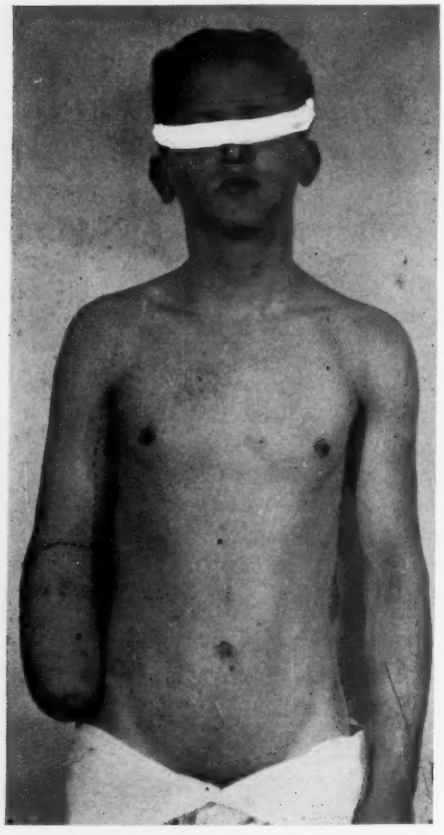


FIG. 2.—C. C., in 1925, age 21: Showing recurrence of the angioma in stump of forearm, amputated four years previously. Note congested club-shaped stump; throbbing and purplish in dependent position; dotted lines indicate enormously dilated brachial vessels.

The conclusion arrived at, at the Mayo Clinic was that the cardiovascular disturbance and angioma of the arm were dependent primordially on a congenital arteriovenous fistula of the subclavian vessels. No further operation or surgical treatment was advised for the time being at the Clinic, and the patient returned to Washington University, St. Louis, in September, 1924, and finished the prescribed four years' course in February, 1925. Realizing that his condition was growing worse (enlargement of the arm stump, dyspnea, palpitation on exertion, *etc.*), he came to New Orleans where I first examined him, February 20, 1925.

Physical Examination.—Without any knowledge of the observations made at the Mayo Clinic, except the patient's statement that his case had been diagnosed as an angioma of the arm and arteriovenous aneurysm of the right subclavian vessels, my attention was immediately directed to the stump of the right forearm, which had been amputated four years previously. The appearance of the amputated limb, in comparison with the normal side, is well shown in Figure 2, which shows the club-shaped end of the stump and cylindric contour of the arm, gradually tapering toward the axilla.

AN ARTERIOVENOUS CAVERNOMA OF THE STUMP EXTENDS TO THE AXILLA AS A PLEXIFORM ANGIOMA

The club-shaped end of the stump and larger size of the right arm (5½ cm. greater at the elbow and 5 cm. at the base of the forearm); the visible, heaving pulsation of the stump and of the whole arm up to its upper third with every beat of the pulse; the dark, congested, cyanotic color of the stump when allowed to hang down (Fig. 2), with the marked reduction in size and pallor of the stump when the arm was elevated, were all signs which prepared the observer for the striking phenomena revealed by palpation and auscultation over the stump, namely, loud systolic murmurs blending with the venous hum, and the thrill which could be heard and felt vibrating under the hand from the terminus of the stump, especially in the neighborhood of the amputation scar, up to the upper third of the arm on the inner side, thence to the level of the axilla. On palpation, the brachial artery was felt to be enormously enlarged, growing larger as it approached the stump, where it was lost in the pulsating, heaving, humming stump of the forearm and above the elbow. The superficial veins of the arm also pulsated and were notably enlarged. The muscles of the forearm and lower third of the arm were incorporated, without anatomic differentiation, in one common mass of tense, pulsating, sponge-like tissue that could be grasped and emptied by a squeeze of the hand, giving the impression that the stump had been transformed into a tumor riddled with cavernous spaces through which the blood circulated in great volume. Even the lower end of the humerus, and the terminal distal stumps of the radius and ulna felt enlarged.

AN ARTERIOVENOUS FISTULA SIMULATED BY AN ENLARGED SUBCLAVIAN ARTERY CLAMPED IN THE GRASP OF THE SCALENUS MUSCLES

Pursuing the investigation further up from the elbow to the axilla, and from the axilla to the supraclavicular triangle, the enlarged and vigorous pulsations of the brachial, axillary and subclavian arteries at once attracted attention by their prominence and vigor. Here again, the very large size of the subclavian artery, from its origin as it emerged from the innominate at the suprasternal notch, was most suggestive of an aneurysmal dilatation. Here again, all signs of an arteriovenous aneurysm which had developed in the amputated stump of the forearm were duplicated with even greater intensity. The systolic murmur could be heard above and below the clavicle and transmitted to the sternum as low as the third right costal cartilage, and with the superficial thrill which could be felt all along the subclavian tract, but with greatest intensity about the crossing of the anterior scalene muscle.

All these signs pointed to an arteriovenous fistula which, seemingly, connected the subclavian artery and vein at about the crossing of the anterior scalene muscle. Against this view, however, was the fact that the enlargement of the artery did not end at the supposed seat of the arteriovenous fistula, but continued, enormously dilated throughout its course, down the arm to the elbow, where it was lost in the spongy mass of the cavernoma at the end of the stump, previously described.

The question that arose was whether the enlarged artery and dilated veins were sufficiently accounted for by the multiple arteriovenous communications in the cavernoma, or was there, in addition, an arteriovenous communication in the subclavian vessels as a part of the congenital vascular pathology of the extremity. Remembering that the enlargement of the artery on the proximal side of an arteriovenous fistula and the increased volume of arterial blood that flows through it is, teleologically speaking, but an effort to compensate for the relative ischemia of the tissues below the fistula, and that the enlargement of the artery is directly proportional to: (1) The size of the fistula; (2) the volume of arterial blood that is short-circuited into the veins at the fistula; and (3) the duration of the fistula; and, since all these conditions were present and even enormously exaggerated in this case, I was inclined to attribute the enlargement of the whole arterial system of the upper extremity to the presence of the

cavernoma. On the other hand, the murmurs and thrill in the upper supraclavicular region seemed to contradict this view in favor of the fistula. I, therefore, decided that the only positive way to clear our doubts was to determine the presence or absence of the fistula by a thorough exposure of the subclavian vessels.

Systemic Cardiovascular Signs and Symptoms.—Before attempting an operation, however, it was decided that the cardiac and systemic cardiovascular changes that had been effected by short-circuiting of the arterial circulation in the cavernoma of the arm had to be carefully studied. To this end, a series of teleoroentgenograms showed that the *heart was enlarged* in all its diameters (Fig. 3). Auscultation revealed the heart sounds to be of normal quality, accelerated but of regular rhythm, accompanied, however, by a

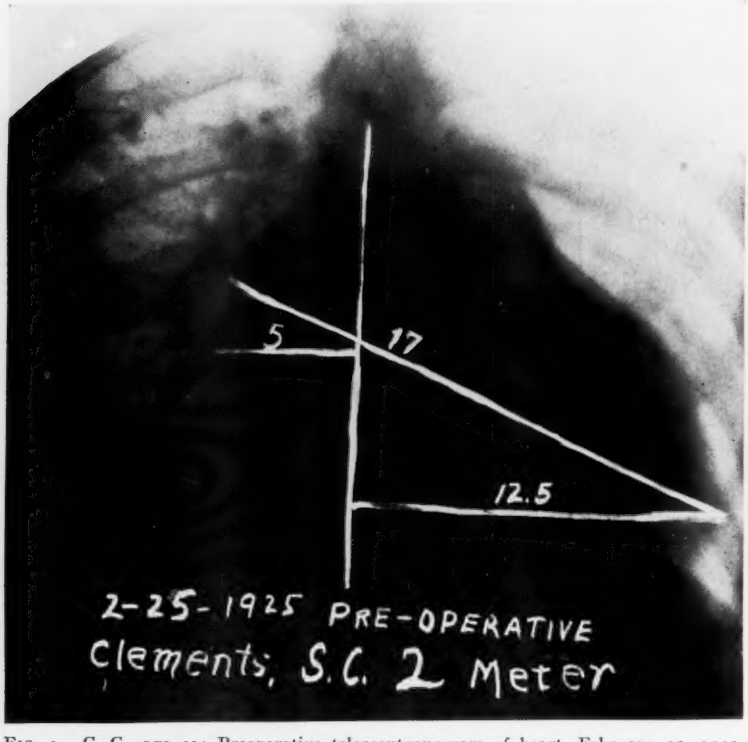


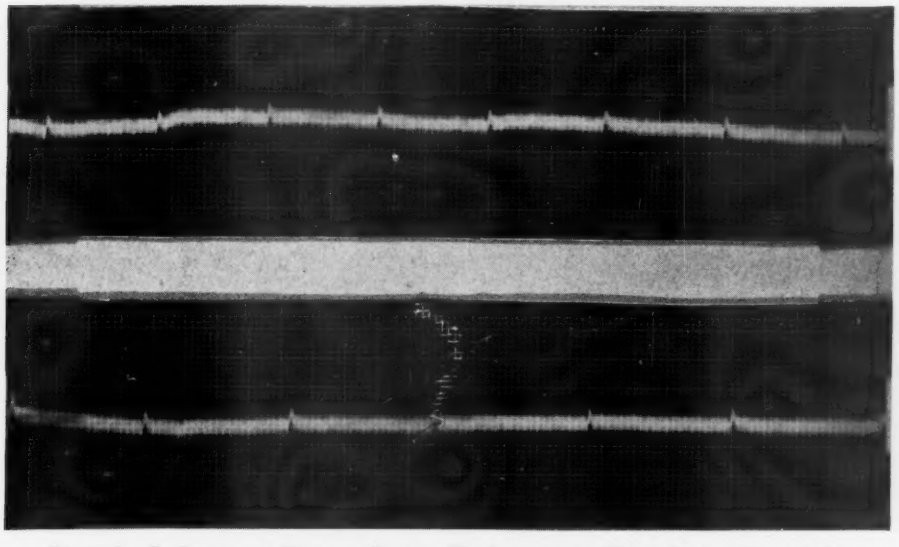
FIG. 3.—C. C., age 21: Preoperative teleroentgenogram of heart, February 25, 1925, before suppression of the angioma by amputation of the arm.

loud systolic murmur which could be heard at the apex, which radiated up to the pulmonary area, and was heard loudest in that area. Fluoroscopically, the cardiac shadow was seen to *contract* appreciably when: (1) Firm digital pressure was put upon the subclavian artery, in the supraclavicular triangle, sufficiently to arrest pulsations and hush the sounds in the cavernoma of the arm; (2) the same result was obtained by encircling the arm with an elastic tourniquet or the sleeve of a sphygmomanometer; and (3) *when the spongy stump of the forearm was squeezed with a firm grip of both hands.* Coincidently with the contraction of the heart shadow, the pulse dropped from 80 to 70, and less, while the blood pressure in the left (normal) arm rose from 102 Mm. to 130/72, which was also the pressure obtained in the angiomatous arm.

The systemic effect of the angioma on the heart and circulation was more accurately and fully confirmed by Professor Garrey's studies of the patient in the Physiologic Laboratory of the Medical School of Tulane University, polygraph records and the electrocardiograms taken by Dr. B. Heninger in charge of the Heart Station at Touro (Graph 1). All these combined to demonstrate the arteriovenous structure of the

angioma and the characteristic effects of arteriovenous fistulae on the heart, the pulse rate and blood pressure—which I have collectively described as the "Branham syndrome,"[23] when they are manifested upon compression or obliteration of an arteriovenous fistula in traumatic arteriovenous aneurysms, and as the "Nicoladon[4]-Israel[5] phenomenon" upon compression of an *arteriovenous angioma*, as in this case. All these signs and symptoms, so typical of the systemic effects of an arteriovenous fistula, made it necessary to intervene promptly in order to stop the arterial leak and the venous plethora that had already exercised a very damaging effect on the heart in this case.

Since compression of the third division of the subclavian artery reproduced the "Branham bradycardiac syndrome" with as much vividness as when the angiomatous stump was compressed by the hand or the cuff of the manometer, I decided to begin the attack on the angioma by exposing the subclavian tract and obliterating the fistula, if



GRAPH I.—C. C., age 21: Electrocardiogram, March 19, 1925: (Heart Station, Touro Infirmary, Dr. B. Heninger), showing effect of compression of the angiomatous arteriovenous stump. Average length of cycle 0.65 sec. Note the immediate slowing of the heart rate. This immediate slowing is at the expense of the diastolic period of the heart cycle (S–T interval). Average length of cycle after compression is 0.85 sec.

this was revealed. If no fistula existed, the artery would be ligated, in the hope that the ligation would check the growth of the angioma. To this end, the patient was admitted to the Touro Infirmary, April 2, 1925, and the operation was performed, April 3, 1925.

Operation.—Preliminary control of the innominate artery for the exploration and search of a suspected arteriovenous fistula, with negative result. Ligation of the subclavian vessels:

Under local and regional (novocain-adrenalin) anesthesia, the innominate artery, greatly enlarged, was exposed as it rose out of the chest into the neck 1 ½ cm. above the upper border of the sternum. The artery was as large as the normal abdominal aorta in its lower infrarenal course and was easily isolated and controlled by an elastic traction ligature (No. 1 soft rubber catheter) placed below the bifurcation of the innominate. Traction on this immediately reduced the size of the arm, stopped the thrill and silenced all the aneurysmal sounds in the neck and in the arm. A temporary ligature on the subclavian at the inner border of the scalenus anticus produced the same effect, showing that there was no arteriovenous fistula anywhere in the course of the subclavian artery, from its origin in the innominate to the crossing of the scalenus anticus. In addition, a very clean and bloodless dissection of the subclavian vessels, artery and vein conclusively demonstrated the total absence of any communication between the vessels; the

thrill and bruits being explained by the *constriction of the enormously enlarged subclavian artery* as it was held in the grip of the scalenus muscles. By dividing the anterior scalenus muscle, the artery was released from the constriction and the aneurysmal noises were silenced, even on relaxation of the ligature. When it was clearly demonstrated in this way that there was no fistula, the subclavian artery was definitely ligated outside of the anterior scalenus muscle, and the provisional traction loop on the innominate artery was removed. The corresponding subclavian and internal jugular veins, both very much dilated, were also ligated near their junction at the innominate bifurcation.

At the close of the operation, the blood pressure in the right arm was found to have dropped to 42 systolic and no definite diastolic. On the normal side the blood pressure was 110/68. The angiomatous arm, itself, appeared to have been the most benefited by the ligation. The stump at the forearm shrank and became noiseless and pulseless, while the pulse slowed and the general blood pressure rose. Cardioroentgenograms, taken three days after the operation, seemed to show a reduction in the midright diameter of the heart.

Return of Pulsation and Relapse of the Angioma.—The patient recovered without shock and the wound healed quickly, but, at the end of a week, the angiomatous stump began to pulsate and, in another week, the arteriovenous signs recurred in the arm, showing that the collateral circulation had been established. With the return of the arteriovenous short-circuit in the arm, the patient suffered from attacks of acute paroxysmal pain in the shoulder which could not be accounted for, except as a neuritis of the acromial branches of the cervical plexus. The persistence of these neuralgic attacks, which required large doses of sedatives for relief, and the increasing evidence of a relapse in the angioma, hastened the decision to amputate at the shoulder joint.

Operation.—The disarticulation at the shoulder joint was performed under ethylene-gas anesthesia, April 24, 1925, 21 days after the ligation of the subclavian, and after complete aseptic healing of the wound. In amputating at the shoulder joint, hemostasis was obtained by carrying an elastic constrictor around the axilla and over the acromion where it was prevented from slipping by a Wyeth pin. The operation was planned to make the arm flap as short and as far away from the angiomatous growth as possible. The axillary contents were cleared—no enlarged or suspicious nodes were discovered; the axillary artery was very large—as large as the normal innominate; and the veins of the arm, superficial and deep, proportionately of large size. The axillary vessels were ligated separately—despite the ligation three weeks previously. While dividing the branches of the axillary plexus, signs of shock suddenly appeared. The blood pressure, which had risen at the beginning of the operation to 158 systolic, suddenly dropped to 118, while the pulse rate rose rapidly from 80 to 152. There had been practically no loss of blood and this sudden shock could not be accounted for, except by the possible effect on the heart of the nerve trauma, or by the sudden drop in the volume of the circulation going through the heart brought about by the amputation of the angiomatous mass. This threatened collapse was averted by a prompt intravenous infusion of hot saline solution, the patient leaving the operating room with a blood pressure of 130/70.

Recovery from the amputation was complicated by a skin infection and later, on the eleventh postoperative day, by a bleeding vein, from which the ligature had been prematurely detached. The bleeding was promptly controlled, but healing was delayed by a hemostatic pack left in the wound for three days, after which the healing progressed rapidly, and the patient was able to leave the hospital, June 11, 1925, about six weeks after the amputation.

Pathologic Anatomy and Histology of the Angiomatous, Amputated Arm.—Gross: Doctor Lanford: An arm, disarticulated at the shoulder joint (Fig. 4), and the same specimen injected with lipiodol (Fig. 5) was radiographed before attempting any dissection. The forearm had been amputated at the middle third five years previously (September, 1920) and the stump was most typical of the lesion. "A cross-section through the forearm below the bend of the elbow, cutting through the radius and ulna, exposes the muscular and other tissue planes all fused together and partly transformed into an

erectile cavernous tissue, in which all the anatomic arteries and veins are seen greatly enlarged. The brachial artery measures 1.5 cm. in diameter, and its walls about 1 Mm. in thickness. The vein measures almost 3 cm. in diameter. A careful examination shows that the soft parts are universally permeated by a coarse network of blood vessels which, in many areas, has replaced the muscular tissue. There is no degenerated, infiltrated and undifferentiated tissue, but a displacement and replacement of the normal tissues by the overgrowth and dilatation of the blood vessels of the part, from the capillaries up, the largest arteries and veins all seemingly intercommunicating through their branches to form the erectile arteriovenous tissue.

"While much of the angiomatous tissue can be accounted for by the simple dilatation of the normal vessels of the part, as the result of the penetration of the arterial stream into the venous circulation, there is also a large, if not the major part of the angiomatous tissue, that is truly neoplastic. This new growth is in the nature of an homeoplastic

FIG. 4.—C. C., age 21: Gross specimen of arm after disarticulation at right shoulder joint, April 24, 1925. Forearm had been amputated at middle third five years previously (September, 1920). A cross-section of the forearm immediately above the stump terminal. Specimen had been preserved in kaiserling several months before section was made. Note the great dilatation of the veins, which in life pulsated like arteries. Throughout the whole section the muscular planes are practically replaced by a coarse reticulum of intercommunicating arteriovenous channels (cavernous erectile tissue). Many of the veins are filled with a clot, hardened by the preserving fluid.

hyperplasia in which new blood vessels are born, and multiply in form, true to type, but with restrictions, which differ from the malignant growths, in the fact that the vascular endothelium here arrives at maturity and does not proliferate indefinitely as an atypical embryonic tissue."

The tumor is, therefore, histologically benign, though in its progressive growth, replacement of the normal tissues, and recurrence after amputation, it presents some aspects of malignancy. The photomicrographs, shown in Figures 6, 7 and 8, exhibit sections through the skin and subcutaneous tissue. Here, the normal, but enormously dilated, arteries and veins are shown and, next to them, innumerable well-lined endothelial blood passages which carry the arterial blood into the venous system, and which constitute the bulk of the erectile growth. It is the fact that the endothelial and connective tissue cells, of the angioma attain full maturity as blood vessels that gives them their benign character.

Repeated fluoroscopic, radiographic, and electrocardiographic studies were made after the amputation, which confirmed the clinical evidence that the heart and circulation had been vastly improved by the operation. The size of the heart was reduced but still remained large (Fig. 9), and "the systolic murmur at the apex radiating upward to the pulmonic area, where it is heard loudest," continued to be heard, but much hushed, as a seemingly permanent relic of the damage created by the primitive arteriovenous fistulae in the angioma. Figure 10 shows the patient as he appeared, January 20, 1926, about nine

months after the amputation. The best proof of the patient's symptomatic improvement was his statement, January 27, 1928, nearly three years after the operation, when he wrote: "During the last four years, I have enjoyed the best of health, my only ailments have been a few colds. I have led a normal life, played golf and bowled for exercise, and have done everything in the way of exercise that I have felt inclined to do." Unfortunately, his splendid prospect for continued health was interrupted, August, 1933, when he contracted a lobar pneumonia which overtaxed the heart, and kept him in a critical state until Sep-

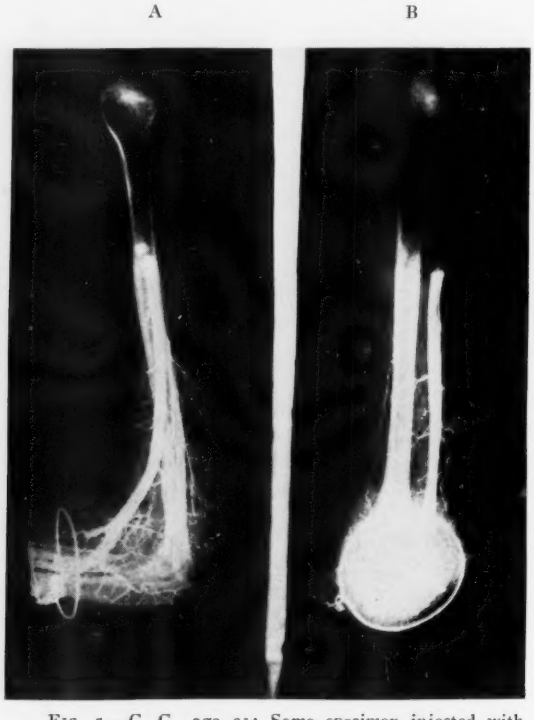


FIG. 5.—C. C., age 21: Same specimen injected with lipiodol. (A) Lateral view: The circle on the distal side of the stump exhibits a wire constrictor tightened around the stump to prevent escape of the lipiodol through the open vessels. (B) Anteroposterior view: Shows enlarged brachial artery lost at the terminus of the stump in the shadow of the tissues obscured by the radiopaque injection. Note atrophy and decalcification of humerus despite increased vascularity of the bone marrow through the enlarged nutrient and periosteal arteries.

Unfortunately for the picture, the terminal end of the stump had been cut off before the lipiodol injection was attempted, and in this way the view of the final merger of the radial and ulnar arteries with the great pulsating arteriovenous caverns at the end of the stump is lost.

tember 15, when he continued to improve. He had fully recovered and resumed all his normal activities when an unsuspected recurrence of the angioma in the axilla, and another at a distance in the chest wall, brought him back to New Orleans, March 24, 1936, for further investigation and advice.

Metastases Appear 11 Years After the Disarticulation.—It would appear, therefore, that a period of nearly 11 years had intervened between the amputation (which, in 1925, had seemingly eradicated the angioma) and reappearance of the angioma in the axillary stump and chest wall early in March, 1936. During these years the patient had been seemingly free from all manifestations of the disease and had no intimation of recurrence until Dr. J. J. Singer, of St. Louis, in the course of a general periodic health examination, made the discovery of the recurrence, which brought the patient back to my clinic, March 24, 1936.

Examination of the patient on arrival fully confirmed the diagnosis of a regional recurrence in the axillary scar of the amputation and another, seemingly a true metastasis, at a distance, in the sixth right anterior intercostal space below and to the inner side of the nipple. These new angiomatous deposits, or formations, presented all the aneurysmal characteristics of the original growth in the arm, namely, a distinct, diffuse pulsation, and a much subdued thrill; a continuous venous hum with systolic reinforcements, also much subdued; all of which ceased on compression of the whole area, only to return when the pressure was removed. The area occupied by the axillary growth is well shown in Figure 11. The angiomatous area presents no recognized boundary line or discoloration of the skin that would indicate its extent. On close inspection, it is seen to fill the upper axilla where the pulsation can be detected by careful palpation over an irregular elliptical

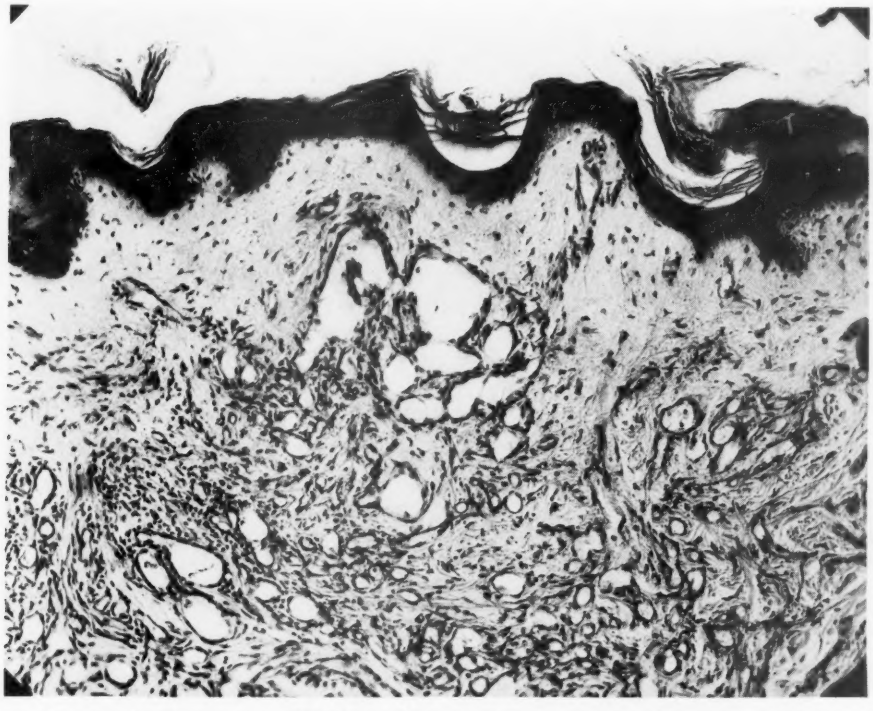


FIG. 6.—C. C., age 21: Photomicrograph of section through the skin of the angiomatous stump showing large cavernous spaces lying in the subcutaneous connective tissue where the larger spaces are in many places divided by trabeculated septa, all lined with typical mature endothelium. As the section penetrates into the muscular layers and beyond the aponeurotic planes, the lacunar spaces become smaller and the muscle tissue is permeated, and largely replaced, by a fine reticular arteriovenous capillary plexus or network. (Hematoxylin-Eosin, ×60) Dr. J. A. Lanford, April 24, 1925.

area 6 or 7 cm. long, distinctly involving the fat and subcutaneous tissue to the same extent.

The center of the angioma, and seeming vortex of the circulatory storm, is well indicated in the painted area in and about the scar left by the disarticulation at the shoulder 11 years previously (Fig. 12). Here the thrill and the pulsations are fairly well defined by palpation, the systolic murmurs and venous hum being distinctly heard over an even wider area with a stethoscope. These bruits, however, are much moderated and softened in comparison with the murmurs that were heard in the arm before the amputation.

The whole bulging area in the armpit, and below it, is quickly flattened and silenced by compression with the hand. An aspirating needle introduced into the most prominent bulge of the tumor immediately filled the barrel of the syringe with bright red blood,

showing that the arterial current largely preponderates in the circulation of the tumor.

In addition to the axillary angioma, a fairly well defined circle of faint pulsation can be detected below and to the inner side of the right nipple, in the area indicated in Figure 14, anterior view. This circular angioma seems to lie deeply under the skin of the intercostal space. There is no discoloration of the skin, or prominence, that would indicate the presence of the tumor. Like the axillary growth, it is an entirely

FIG. 7.—C. C., age 21: Section of the same tumor through the muscle tissue showing enormously dilated venous channel. (×60) Dr. J. A. Lanford, April 24, 1925.

subcutaneous formation which lies between the skin and the musculo-aponeurotic planes. In this connection, we realize that these neoplasms are disconnected, the compression and obliteration of the axillary growth having no effect on the thrill, pulsations and murmurs of the intercostal angioma.

In order to explore the interior of the thorax for other possible metastases and determine, if possible, any connection existing between the axillary and intercostal growths, a series of teleoroentgenograms were made (Figs. 13, 14 and 15) which show several views during and after the injection of the two angiomata with thorotrast.

From these we judge that the injectable vessels or spaces in each tumor were relatively small in comparison with the vascular areas recognized by palpation and auscultation, and that they did not extend deeply into the thorax, but were seemingly confined

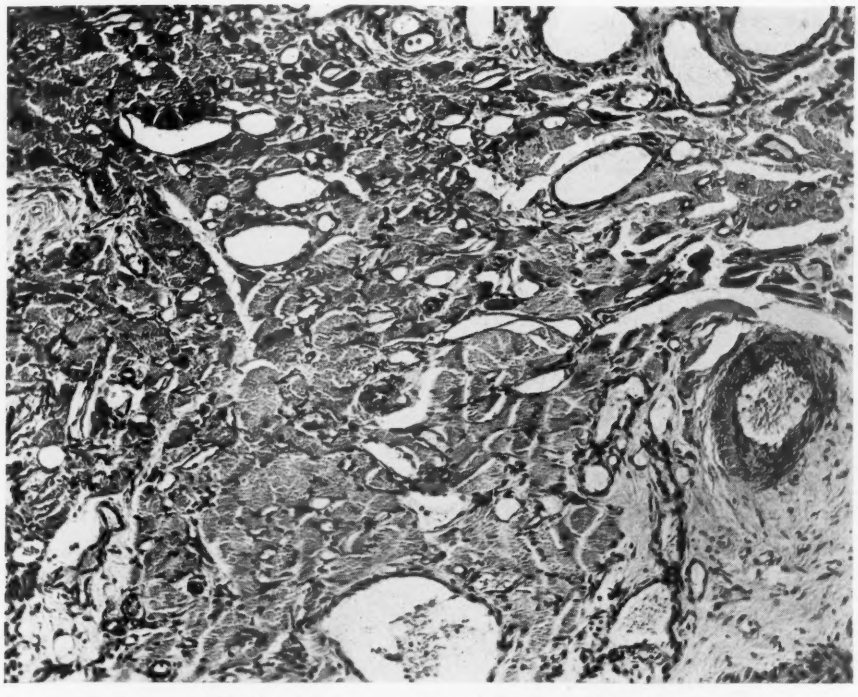


FIG. 8.—C. C., age 21: Section of same tumor. (×240) Dr. J. A. Lanford, April 24, 1925. Note permeation of muscle tissues by dilated blood vessels typical of arteriovenous cavernous angiomata.

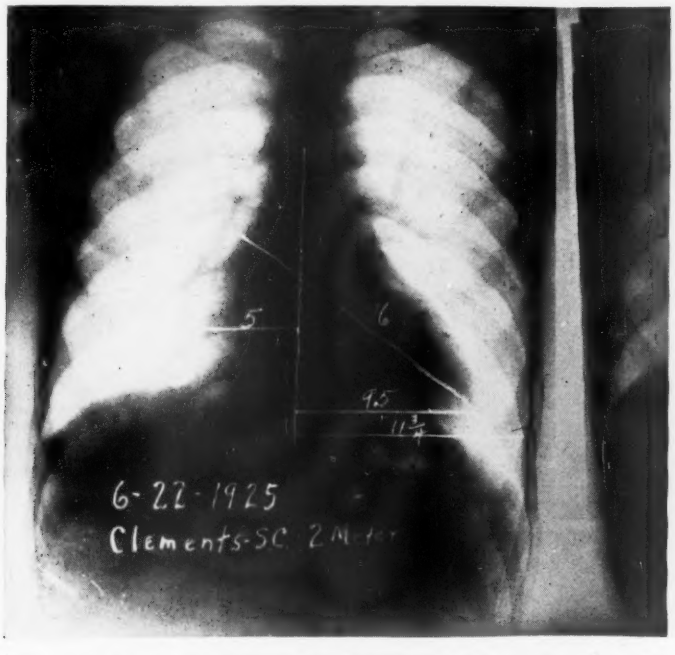


FIG. 9.—C. C., age 21: Postoperative teleroentgenogram of heart, June 22, 1925, two months after amputation of arm.

to the chest wall. The roentgenograms also clearly demonstrated that the two growths were separate and independent of each other. Furthermore, as far as the fluoroscope and films could show, there were no recognizable metastatic deposits in the lungs, mediastinum or heart. The two parietal metastases seem to have been confined to the right half of the chest wall. In considering the practicability of their surgical extirpation, it was also evident that the vascular areas exhibited by the radiopaque injections (thorotrast) represented only the core or vortex of the angiomata, *i.e.*, where the intercom-

FIG. 10.—C. C., age 22; January 20, 1926: About eight months after the amputation at the shoulder joint, April 24, 1925, the shallow, horseshoe-shaped shadow over the right sternoclavicular joint indicates the scar of the incision for the exposure and ligation of the subclavian artery and vein (April 3, 1925). The patient had been completely relieved of his cardiocirculatory symptoms when this picture was taken.

municating channels between the neoplastic arteries and veins were largest and most penetrable to the radiopaque injections. There is no doubt that, beyond these plainly visible central arteriovenous sinuses, there was a widely distributed zone of new or abnormally dilated capillaries which gradually merged with the normal vascular supply of the region.

Preliminary Preoperative Considerations.—In conformity with this view of the angiomata, the area to be excised would have to be much larger than that defined by the radiopaque injections. *The prophylactic control of hemorrhage* in the course of the extirpation was, therefore, a matter that gave serious concern, with the added prospect that, in dealing with the intercostal tumor, the excision of the tumor would be complicated by a pneumothorax.

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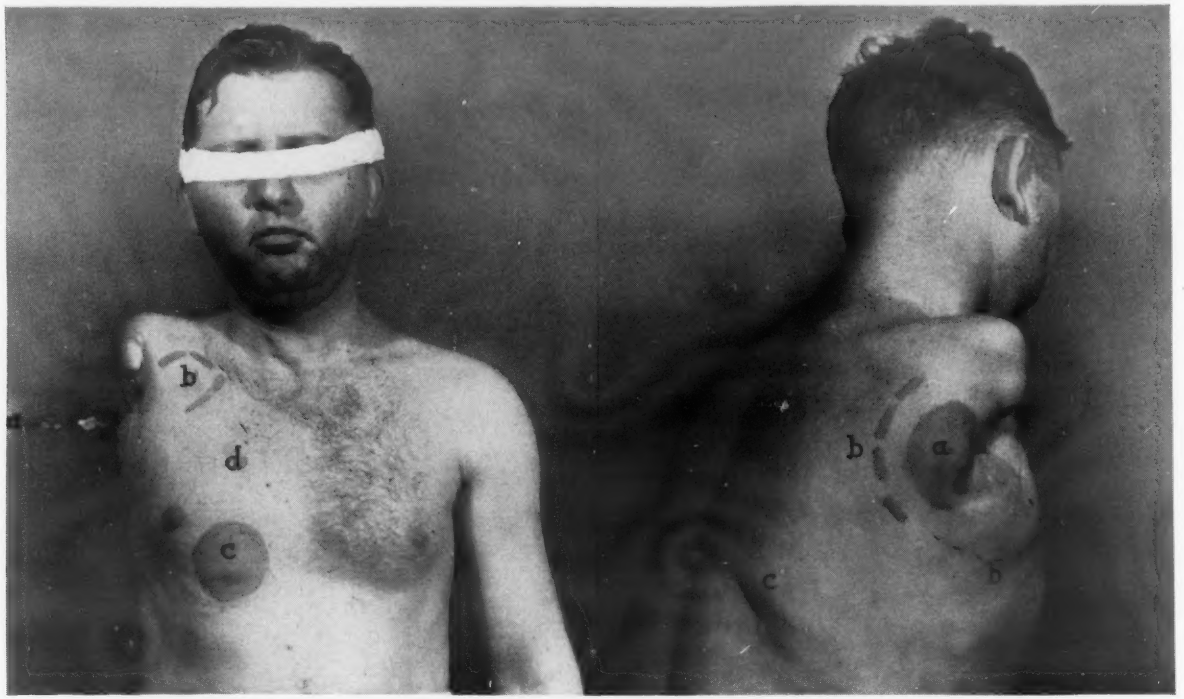

FIG. 11.—C. C., age 32; March 26, 1936: Secondary arteriovenous cavernoma of axilla and chest wall, 11 years after disarticulation of the shoulder for same lesion in the arm.

(a) Needle is seen penetrating the most prominent bulge of the angiomatous tumor in the axilla—about to inject thorotrast.

(b) Interrupted lines seen in anterior wall of chest in the subcoracoid region indicate limits of the arteriovenous murmurs which gradually diminished in intensity up to this level, where they ceased sharply. Above this painted line is the scar indicating where the subclavian had been ligated 11 years before.

(c) The dark circular area below and to the inner side of the right nipple was produced by painting the surface with merthiolate (radiopaque) solution. The bulge of the pulsating angioma is seen in the center of this area, where loud arteriovenous murmurs can be hear with greatest intensity.

(d) Note the total absence of hair on the right half of the chest, apparently a congenital defect.

FIG. 12.—C. C., age 32: (a) Greatest bulge of cavernous arteriovenous angioma in the axilla in the center of the circular area painted with merthiolate. This is the seat of the most intense arteriovenous murmur and venous hum.

(b) The interrupted lines indicate the limit of the audible murmurs. This whole area was excised, April 8, 1936.

(c) The scar in the posterior sixth right interspace is the relic of a thoracotomy for empyema when patient was three years old.

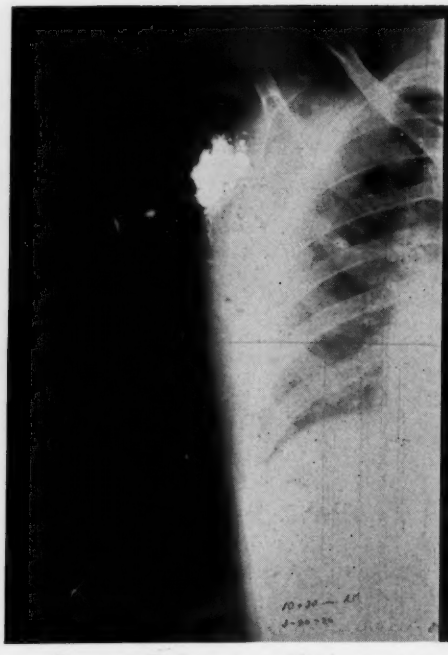

FIG. 13.—C. C., age 32, March 26, 1936: Radiograph showing injection of thorotrast in secondary axillary angioma.

FIG. 14.—C. C., age 32, March 26, 1936: Radiograph showing metastatic angioma in the sixth interspace below the nipple, five minutes after injection with thorotrast. The axillary growth, seen as the opaque injection, is fading away.

FIG. 15.—C. C., age 32, March 26, 1936: Profile view of the intercostal growth in the anterior chest wall, two minutes after injection with thorotrast.

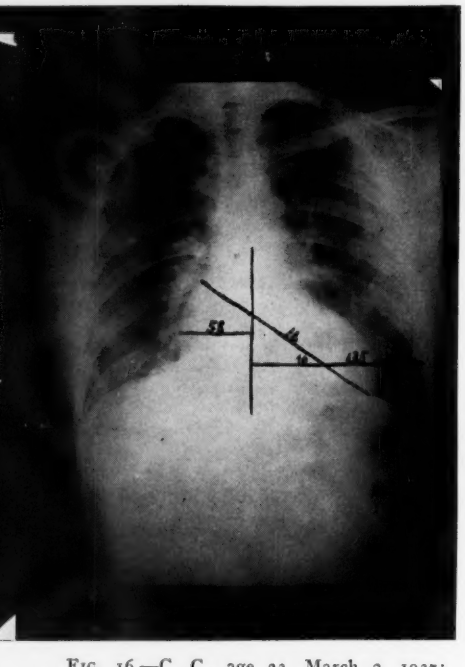

FIG. 16.—C. C., age 33, March 2, 1937: teleroentgenogram of heart, 14 months after extirpation of axillary angioma.

As the axillary growth was the larger, more active, and beginning to thin the skin covering, it was more liable to accidental hemorrhage from trauma, ulceration or spontaneous rupture. It was, therefore, decided to attack this growth first, and reserve the extirpation of the intercostal mass for a secondary operation after the patient's recovery from the first had been assured.

In connection with the roentgenologic studies of the angiomatous growths, several

FIG. 17.—C. C., age 32, March 24, 1936: Radiograph of heart (two meter distance) 15 days before extirpation of metastatic angioma in the axilla. Also shows separate intercostal growth enclosed in the circle.

teleoroentgenograms were made of the thoracic contents, especially of the heart and aorta. Figure 16 gives an anteroposterior view of the chest which shows that the dimensions of the heart have slightly increased since the last roentgenogram taken in 1936 (Fig. 17) when it was found that the heart contracted very noticeably after the amputation of the arm. Observations, made at this time, showed that sustained compression of the angiomatous area lowered the pulse rate from 85 to 80 and increased the blood pressure in the arm by a few millimeters—without compression, pulse rate 85, blood pressure 138/70; with compression, pulse 80; blood pressure 140/72. The bradycardiac phenomena of an arteriovenous fistula under compression were present here (Branham syndrome; Nicoladoni-Israel phenomenon) but much less pronounced than when the cavernoma of the forearm was compressed before the amputation of 1925. Corresponding evidence of slight but positive increase in the diastolic interval under compression was also furnished by the electrocardiogram.

Extirpation of Axillary Metastatic Angioma.—In the absence of all serious contraindications, the patient was admitted to the Touro Infirmary, April 7, 1936, and the operation was performed the next morning under combined local and general anesthesia. After

a preliminary sedation with nembutal, followed in one hour by a hypodermic of morphine and scopolamine, the skin covering the central core of the angioma was anesthetized with novocain-adrenal solution. After this, the whole pulsating area was densely infiltrated with ephedrine sulphate (1:1,000) in physiologic salt solution. This was injected directly into the blood-filled cavernous spaces of the central core in order to obtain the maximum diffusion and vasoconstrictor effect of the drug on the tissues. The whole peri-angiomatous area was edematized and made hard, pale, ischemic, and pulseless with the same solution injected with a Dunn infiltrator. In this way an elliptic area 15x7 cm. was thoroughly

FIG. 18.—C. C., age 32: Section of axillary angioma extirpated April 8, 1936. (×60, H and E stain) Dr. J. A. Lanford.

edematized and rendered ischemic and pulseless to the depth of the musculo-aponeurotic planes, thereby completely circumscribing and enveloping the angioma with a vaso-constricting atmosphere which made it possible to attack the tumor in a relatively blood-less zone at the periphery. At this stage the anesthesia was continued with cyclo-propane-oxygen gas.

The excision was begun by a curvilinear incision which extended about two-thirds of the circumference of the ellipse which enclosed the angioma (Fig. 12). Despite the marked ischemia that had been obtained by the injection, much time was consumed in controlling numerous vessels which were encountered as the tumor, in solid block, with the skin covering and fat, was lifted out of its deep axillary bed to the depth of the latissimus dorsi and the serratus muscles, which were exposed. After complete hemostasis was obtained by clamps, ligations and electrocoagulation, the excision of the mass *in toto* was effected by completing the elliptic outline of the incision. The solid block of tissue thus excised contained all the recognizable elements of the tumor, including practically all the ephedrine solution (over 250 cc.) which had been injected for the benefit of its ischemic effect.

It is worthy of note that the arteriolar and capillary system in the vicinity of the

angioma showed an increased volume and capacity as the vessels converged centripetally to the central core of the cavernoma, where the arteriovenous channels or sinuses met, as in a confluent, to form the vortex of the angioma.

The excision of the angiomatous block left quite a gap in the anterior axilla-pectoral region, but this was readily filled and closed by undermining and mobilizing the walls of the cavity, and holding the cutaneous edges in place with interrupted silk and dermal sutures. The wound was supported with broad adhesive strips and a copious sterile dressing under a Desault bandage.

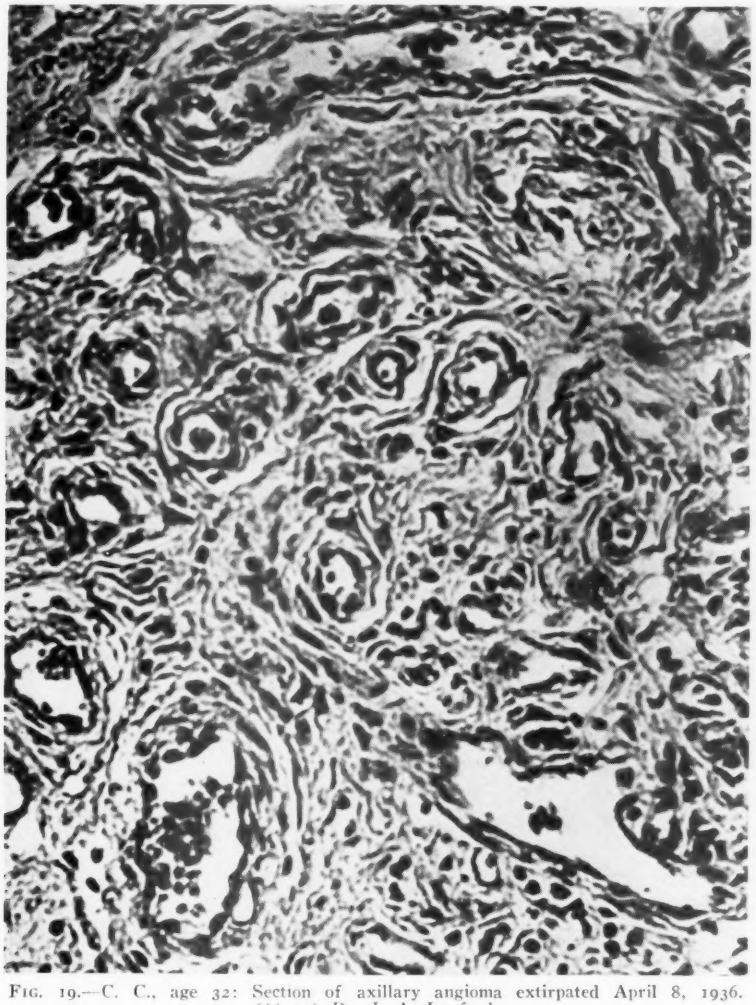

FIG. 19.—C. C., age 32: Section of axillary angioma extirpated April 8, 1936. (×240) Dr. J. A. Lanford.

It should be noted that, despite the great care to reduce the bleeding to a minimum by the prophylactic hemostasis previously described, the blood pressure began to fall and the pulse to quicken while the tumor was being excised from its deeper attachments. Without waiting for further evidences of shock, a glucose-saline drip was instituted through the internal saphenous vein at the ankle, and continued throughout the operation until the patient was put to bed and fully restored.

Pathologic Examination.—Gross: Dr. J. A. Lanford. "Specimen consists of an hemangioma removed from the right axillary region. It is covered by an elliptical piece of skin measuring 11½x6 cm. The periphery of the skin shows some scar tissue in a limted

area, but most of it is normal in appearance. The under surface of the mass measures 12x6½x5 cm., and is made up mostly of adipose tissue which had previously been cut through, showing many large and small blood vessels coursing through the fat, and all converging towards a center nearer the skin surface where the vessels united to form the cavernous areas of the hemangioma.

"A photomicrograph made from a section of the same specimen (Fig. 18) shows an irregularly diffused infiltration of the subcutaneous fat by a network of capillaries, which tend to converge, without definite limitations towards a common center where the vessels merge to constitute an arteriovenous confluent or vortex. The lymph nodes of the same region are free from metastatic deposits, though the capsules appear to be involved in the angiomatous process.

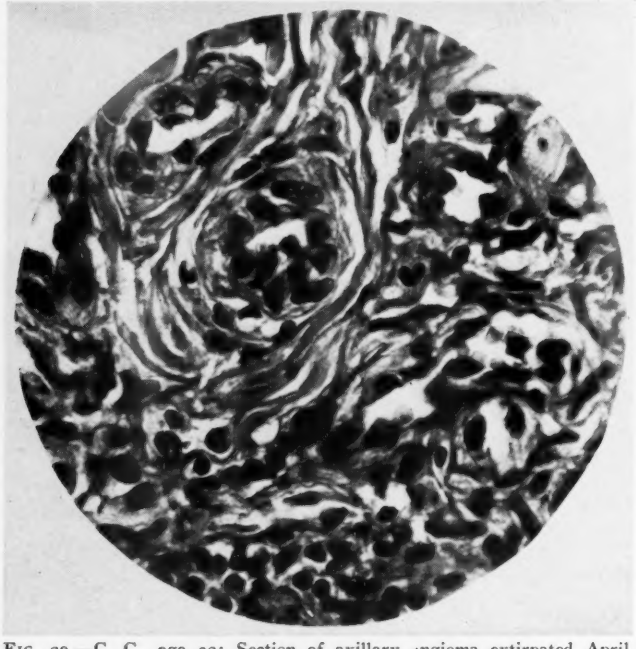

FIG. 20.—C. C., age 32: Section of axillary angioma extirpated April 8, 1936. (×360) Dr. J. A. Lanford.

"The same specimen, examined with higher powers (×240 [Fig. 19] and ×360 [Fig. 20]), shows a connective tissue stroma of a reticulo-endothelial type with a large preponderance of endothelial cells, enclosing numerous blood channels or sinuses of new formation and variable size, lined by well differentiated endothelial cells arranged in cylinders of the same pattern, mostly held together by an outer layer of concentrically arranged spindle-shaped cells. In places, the septa which separate these channels have broken down, thereby creating large cavities or cavernous blood spaces. It is in this respect that this metastatic growth differs from the original parent angioma in the arm (Figs. 6 and 8), for in that tissue the normal anatomic vessels, whether arteries or veins, very much dilated from the effect of the congenital arteriovenous fistula, developed into pulsating blood spaces, which are grossly recognized as cavernomata. It was also the preponderance of blood vessels of new formation that gave this metastatic growth a more neoplastic character than that recognized in the parent tumor, which developed largely on a ground-work of preformed vessels."

Subsequent Course.—The extensive wound left by the operation, though free from infection, healed slowly. The edges of the wound gave way at the end of one week, leaving an unhealthy, granular surface which bled at the slightest provocation. On one occasion, it became necessary to apply a gauze pack soaked in spirits of turpentine in order

to control the persistent ooze from the granulations. Repeated attacks of intermittent fever, without visceral complications, occurred during the convalescence, which were apparently controlled by quinine, despite the absence of plasmodia from the blood. These attacks prolonged the convalescence until the end of May, 1936, when the wound spontaneously assumed a healthier aspect and began to heal rapidly. While the wound was still being dressed, the patient returned to his home, June 12, 1936.

Roentgenotherapy.—After the patient returned to St. Louis, in June, 1936, his wound healed progressively, and by the middle of July, 1936, he was entirely well and had returned to his usual occupation. As planned before his departure from New Orleans, he was given a course of roentgenotherapy, at Barnes Hospital, under the direction of Drs. J. J. Singer and S. Moore. The radiations were applied for the double purpose of: (1) Preventing, if possible, a recurrence of the angioma in the excised area; and (2) controlling and obliterating the angiomatous patch below the right breast, which had remained untouched, except for the injection of thorotrast given for diagnostic purposes, March 26, 1936.

The radiations were applied, successfully, to three well-defined areas (mammary A1, axillary A2, and subscapular A3) in accordance with the following formula:

	Area	Portal Sq. Cm.	K. V.	Filter	Dist.	A. U.	M. A.	Min.	Per Cent of Depth Dose	R. {Ma. / Min.	Total R.
10/15/36.....	A1	225	200	0.5 cu. 1 Al.	50 cm.	0.13	15	9.2	35	—	550
10/16/36.....	A2	225	200	0.5 cu. 1 Al.	50 cm.	0.13	15	9.2	35	—	550
10/17/36.....	A3	150	200	0.5 cu. 1 Al.	50 cm.	0.13	15	9.2	35	—	550

A light erythema reaction followed this application. Nothing further was done until August, 1937, when, on a visit to New Orleans, another series of radiations were applied at the Touro Infirmary for three successive days (250 R. each; 750 in all) and were concentrated on the circular submammary patch (which had never been operated upon) in accordance with the following formula:

	M. A.	K. V. P.	S. T. D.	Filter	R.	Area
8/5/37	20	140	50	0.25 cu. 1 Al.	250	1
8/6/37	20	140	50	0.25 cu. 1 Al.	250	1
8/7/37	20	140	50	0.25 cu. 1 Al.	250	1

A very slight erythema followed this application. The patient continued to do so well, physically, that he married, October 1, 1937 (17 months after the excision of the axillary angioma), and on October 29, 1939, his first child, "a fine, lusty, baby girl," was born without any apparent physical defects. He had been in good health and active during the nearly four years that followed the extirpation of the axillary metastases. He had suffered only from a very transitory attack of lumbar pain which was diagnosed renal colic, without demonstrable calculi.

At my request, the patient has had himself examined by his family physician (Dr. H. D. Spector), who is familiar with his history and who reported, November 30, 1939, that the patient was in excellent general condition, active and working very satisfactorily at his business (furniture store); well-nourished, weighing 145 pounds—a little overweight; pulse 80–90, regular; blood pressure 120/80, temperature normal, and the laboratory tests for blood and urine revealed no abnormalities. Physical examination of the chest revealed some moisture (mucous râles?) at the right base, with no clinical symptoms, however. There was some tenderness and induration of the chest wall at the site of the old submammary angioma, where the thorotrast had been injected four years previously. Apart from this, there were no pulsations, thrills or murmurs to indicate a persistence or recurrence of the angioma in the chest wall. Physical examination of

the chest and abdomen failed to reveal any demonstrable tumors or metastatic deposits.

The photograph of the patient (Fig. 21), December 1, 1939, confirmed the report of his good health. The teleradiograph of the heart and chest (Fig. 22) shows a slight increase in the oblique longitudinal axis (right auricle to apex) as compared with the last radiograph (Fig. 16).

The notable fact in this patient's cardiac history is that the heart has never enlarged to the great size it attained in 1925, before the disarticulation of the arm with the angioma (Fig. 3), or before the excision of the secondary axillary angioma, in 1936 (Fig. 9*).

FIG. 21.—C. C., age 35, December 1, 1939: Note congenital absence of all hair in right half of chest.

Discussion.—The hemangiomata have been classified from the viewpoint of the metastasizing tumors of this class, into the following groups:

(1) Definitely malignant metastasizing hemangiomata (hemangio-endotheliomata, angiosarcomata, *etc.*) in which the secondary growths are also, histologically, malignant. Numerous illustrations of this group are quoted by Sonntag,[7] Wright,[8] Robinson and Castleman,[9] Geschickter and Keasbey,[10] Matas,[11] and others.

(2) Metastasizing hemangiomata, in which the primary and secondary tumors are seemingly, histologically, benign, the malignancy being recognized clinically, chiefly by the metastatic features; originally a very rare practically unknown group, but now forming a growing list since the first cases were

*For more details regarding the remarkable cardiologic features in this patient's case, see paper by Dr. B. R. Heninger and the author.[6]

reported by Homans,[12] Boormann,[13] Shennan,[14] Ewing,[15] Sonntag[16] and, later, by Robinson and Castleman,[17] Livingston and Klemperer,[18] Ward and Jones,[19] Hall,[20] Abrams, *et al.*[21]

(3) Multiple (nonmetastatic) benign hemangiomata appearing contemporaneously over the body, but particularly in the abdominal visceral organs (liver, spleen, *etc.*) (Sonntag,[22] Wollstein,[23] Taylor and Moore,[24] Matas,[25] Jaffé,[26] and others).

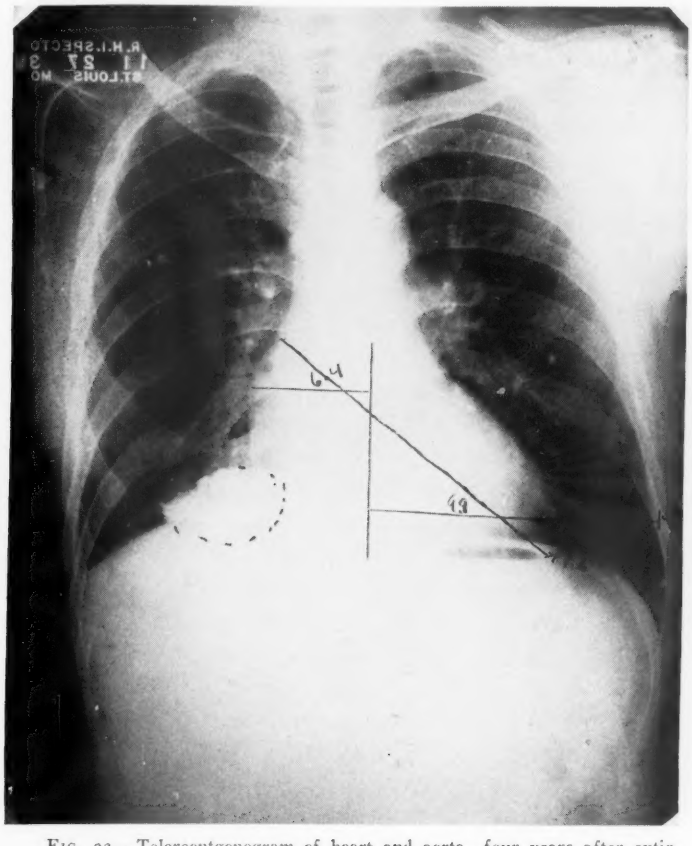

FIG. 22.—Teleroentgenogram of heart and aorta—four years after extirpation of axillary angioma. The dotted outline of circle on the right of heart shadow indicates the seat of the inactive secondary submammary angioma which still retains thorotrast injected four years previously.

In the case here reported, we have presented a histologically benign hemangioma of the arm which continued to grow from birth, as an arteriovenous cavernoma from the hand to the axilla, for *21 years*, without any apparent metastasis during that period. At the age of 21, the arm was amputated at the shoulder, the patient recovering and remaining entirely well for 11 years. Then two secondary angiomata, of the same histologic type, made their appearance, one in the right axilla, in and about the scar left by the disarticulation, and the other at a distance in the chest wall, in the right submammary region. Clinically and histologically, the two secondary growths were identical with the primary growth as pulsating arteriovenous erectile cavernomata.

The danger of hemorrhage from rupture of one of the caverns compelled

the extirpation of the larger axillary growth. The other, smaller, less active, but deeper in the chest wall, was allowed to remain unmolested, except for two radiations (roentgenotherapy) within a year after the extirpation of the major growth. Thus far, to the date of this report (December 17, 1939), four years have elapsed since the major (axillary) growth was excised, and the patient has remained well, robust and active, without any signs of recurrence or metastases elsewhere in the body, while the smaller, isolated submammary growth has lost its pulsating character and is undergoing atrophic and obliterative changes.

In view of these facts, the question arises whether these secondary growths are really metastases in the true migratory sense of malignant neoplasms or caused by the tardy or postponed development of aberrant, ectopic endothelial cell nests which ultimately develop and come to the surface, as distinct tumors. The old question, "What is it that makes a seemingly typical 'histologically benign angioma,' occasionally and without known reason, suddenly or gradually assume a malignant behavior, which makes it reproduce itself by metastasis?" has never been answered.

It is indeed surprising, as remarked by Shennan long ago, "that despite the fact that innumerable endothelial cells, in all stages of development, must be carried off in the extremely strong and rapid current of an arteriovenous angioma—they are so rarely metastatic, even though the endothelial cells, wherever they are carried, still remain bathed in their normal nutritive fluid."

The increasing frequency with which metastasizing angiomata are being reported would suggest that the capacity of a structurally benign angioma to reproduce itself is not a conclusive criterion of malignancy.

The fact that in this case, metastases did not occur during the 21 years that the angioma flourished in its most active and aggressive form, and that it was not until 11 years after its extirpation that they made their appearance, is a challenge to all the theories clinically invoked to account for the long-delayed appearance of the secondary tumors as metastases, or for their *de novo* appearance as original tumors of separate and independent origin.

Dismissing, as idle, all further theoretic discussions on the nature and pathology of these secondary metastatic angiomata, we regard the mere statement of facts in this very unusual case as worthy of record, if only as an encouragement to others who may be faced with a prognostic problem of the same initial gravity which has evolved into a later cheerful outlook.

NOTE.—While this article is going through the press, Dr. Spector writes that, on March 11, 1940, Mr. C. C. was reexamined, with special emphasis on evidence of persistence or recurrence of angiomatous signs in the areas previously involved in the metastases. The examination only confirmed the patient's good health and normal activities and complete absence of angiomatous lesions in the chest or elsewhere, as stated in the previous report, above quoted. The cardiologic tests, including electrocardiograms, teleoroentgenograms, etc., show no notable changes since the report of December 17, 1939. In view of the total suppression of pulsation, thrill and murmurs in the unexcised angiomatous area injected with thorotrast (which has remained unabsorbed in the tissues since it was first injected,—over four years) and of the systematic roentgenotherapy to which the angiomatous area was subsequently subjected—it is a question whether the

apparent cure has been obtained by inducing an obliterative vascularitis in the angioma through the separate or combined efforts of the thorotrast and roentgenotherapy, or by some other unknown factor.

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CASE OF LARGE FALSE ANEURYSM OF THE RIGHT SUBCLAVIAN ARTERY SUCCESSFULLY TREATED BY A MODIFICATION OF THE MATAS OPERATION *

WILLIS D. GATCH, M.D.

INDIANAPOLIS, IND.

THIS CASE is reported because of the method of its treatment, which, I hope, may make possible the cure of some aneurysms with a blood supply uncontrollable by tourniquet or ligature.

Case Report.—R. P., white, male, age 41, was admitted to the Indiana University Hospitals, February 28, 1939, for a large aneurysm of the right side of his neck. He complained also of weakness, loss of sleep and shortness of breath on slight exertion.

In April, 1921, a 38 caliber bullet had entered the midline of his neck, two inches above the suprasternal notch, and had lodged in his right axilla. A small swelling appeared at once above his right clavicle. In a week he had recovered sufficiently to visit the Mayo Clinic. Since he was there given no suggestion on treatment, he apparently had no aneurysm at that time. On his return home, the supraclavicular swelling began to pulsate, and six weeks after the injury his right axillary artery was ligated. Following this his entire right arm and forearm were paralyzed, and remained so for about seven months, when a partial recovery began.

During the next 17 years he complained of shortness of breath, which made him unable to walk more than two or three blocks without resting. During this time the lump above his right clavicle did not bother him or change in size.

In July, 1938, he had a severe attack of heart failure, attended by seven weeks of orthopnea and cough. In October, 1938, he had bilateral pneumonia and developed a marked edema of his entire body. During this illness, late in October, 1938, after severe coughing, the pulsating swelling appeared, and reached, almost overnight, the size seen on admission. The mass had not increased greatly in size during the four months since its appearance. The remainder of the history is irrelevant.

Physical Examination.—The patient was powerfully built and well nourished. The right pupil was contracted and the right palpebral fissure narrowed. The veins of the right arm and right side of head and neck were not dilated. There was marked atrophy and weakness of the right arm and forearm.

Dr. George Bond, after an examination of the cardiovascular system, reported considerable enlargement of the heart, hypertension and mild cardiac decompensation. He found the blood pressure in the left arm to be 165/110 and in the right arm 120/110.

The usual laboratory examinations showed nothing abnormal. All tests for syphilis were negative. The aneurysm was the size of a large grapefruit (Fig. 1), and pushed the patient's head to the left. It extended from the midline in front to the anterior edge of the trapezius muscle. It overhung the inner half of the clavicle and the right sternoclavicular joint, and extended upward almost to the jaw. It displaced the trachea about two inches to the left (Fig. 2). The skin over it was black and shining and seemed in imminent danger of rupture at each violent expansion of the mass. The antero-inferior portion of the aneurysm was firm to the touch, the other portions, resilient. No thrill was felt except along a line extending from the suprasternal notch along the internal surface of the aneurysm. We ascribed this finding to compression of the common

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carotid artery. A very loud systolic bruit was heard over the entire swelling. Roentgenologic examination showed that the aneurysm did not extend below the first rib.

The foregoing data seemed to warrant the following conclusions:

(1) The aneurysm arose from either the first or second divisions of the subclavian artery, possibly from both.

(2) Ligation of the first portion of the subclavian artery or of its branches would be perilous or impossible, because of the location of the aneurysm and its long duration. Ligation of the innominate artery would, at best, only diminish the blood entering the aneurysm.

(3) There was but one opening into the aneurysm and this was probably large.

No act of a surgeon is considered more foolhardy than to cut into a live aneurysm, but I decided to do it because I had no other choice. The operation was planned as follows: (1) Constriction of the innominate artery; (2) wide incision of the aneurysm, and digital occlusion of its opening into the subclavian artery; (3) intrasaccular closure of the opening; (4) preparation for massive blood transfusion.

I am very grateful to Dr. John Owen for his able assistance in the operation.

Operation.—March 7, 1939: Fifteen hundred cubic centimeters of blood was obtained from three donors, and preparations were made for its immediate injection when needed, by way of a vein in the right leg. Under ether anesthesia the incision was started over the anterior edge of the left sternocleidomastoid muscle, three inches from the sternum, and extended downward over the left sternoclavicular joint, thence diagonally across the body of the manubrium and downward a short distance along its right border. The left sternoclavicular joint was excised. The manubrium was rongeured away from above downward and from left to right until a good-sized opening was obtained. The ribbon muscles were divided. The innominate artery, the diameter of an index finger of average size, was freed, and a ligature passed around it on an aneurysm needle. The assistant now constricted the artery by pinching the ligature with the thumb and index finger of his left hand. This stopped pulsation of the aneurysm, but did not diminish its size or tenseness.

A second incision about four inches long was now made over the most prominent portion of the aneurysm and parallel to the clavicle. The assistant instantly thrust his right hand into the aneurysm through the escaping blood and plugged the opening in the subclavian artery with his finger. The opening was about one and one-half inches long and a little less than half an inch wide. It was situated just above the inner end of the clavicle. I removed some adherent masses of clot and examined the walls of the aneurysm. The edges of the opening were thin and the bottom of the sac for a considerable distance around the opening was of cartilaginous hardness. It was evident that the opening could not be closed by suture, and that ligation of the artery through the sac wall could not be accomplished.

After a period of anxious thought, I decided that the only possible way to close the opening was to plug it with a piece of the sac wall. This I did as follows: First, I separated the sac from the skin and subcutaneous tissues as far as was easily possible. Since the peripheral half of the sac was necrotic, I cut it away. By sharp dissection I next freed the lateral and inferior portion of the sac, where there was no danger of injury to important structures, down to the upper border of the clavicle. I next fashioned a flap from this portion of the aneurysmal wall, as shown in Figure 3-1. The base or hinge of this flap was located at the upper border of the clavicle. The flap was about three and one-half inches long. It consisted, of course, of condensed connective tissue and was a little less than a quarter of an inch thick.

I now placed three sutures across the finger of my assistant as follows. With a short, full curved needle I took a good bite of the sac as close to the opening as possible, then a like bite on the other side. One suture was so placed at each end of the opening, and one at the middle (Fig. 3-1). I now passed a hemostat beneath the central portions of these sutures, seized the apex of the flap (Fig. 3-2) and pulled

FIG. 1—Patient on admission.

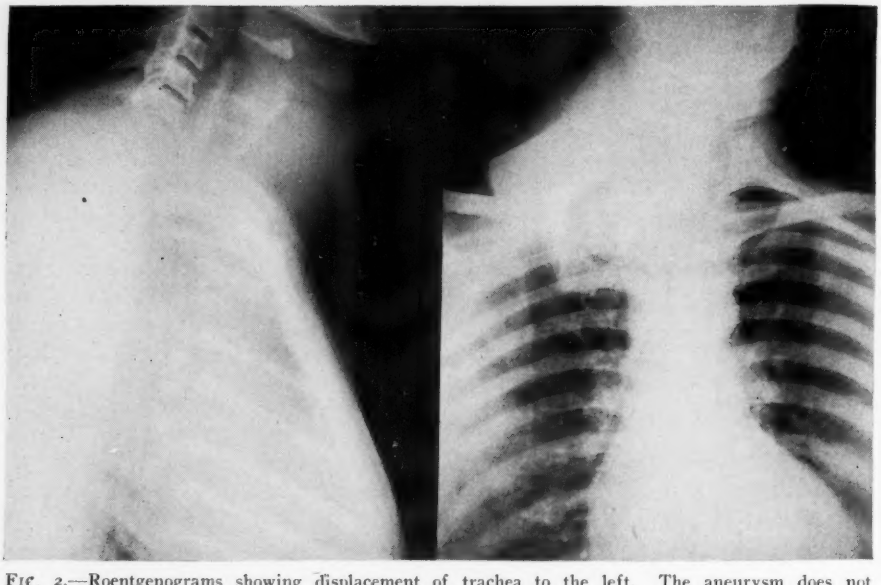

FIG. 2.—Roentgenograms showing displacement of trachea to the left. The aneurysm does not extend below the first rib.

it over the obstructing finger, which was then withdrawn. There was a quick gush of blood which was stopped by packing the flap into the opening where it was held by tying the sutures over it. The flap was further secured by a number of interrupted sutures which fastened its edges to the adjoining wall of the sac. We now freed the remainder of the distal part of the sac from the surrounding tissues, taking care not to free its base where important structures might be injured. The freed portions were now trimmed and cut so that they could be firmly imbricated by sutures over the flap (Fig. 3-3).

The ligature was now removed from the innominate artery, and the wounds closed without drainage (Fig. 3-4). The dressing was strapped firmly over the site of the aneurysm.

While the wounds were being closed the patient was given 600 cc. of blood. His pulse rate at the beginning of the operation was 96, and his blood pressure 170/110; at the close, his pulse was 92, and his blood pressure 118/60. The loss of blood, except for that in the aneurysm, was not great.

Postoperative Course.—Convalescence was satisfactory. The dressing was first changed on the ninth day, at which time primary healing had occurred. No bruit could be heard at the site of the aneurysm.

On the twelfth day after operation, shortly after getting out of bed for the first time, the patient had severe precordial pain and shortness of breath. His blood pressure at the onset of this attack was 130/95, but in three hours it had fallen to 85/50. His condition gave us much anxiety for several days. Dr. George Bond, who attended him at this time, made a diagnosis of acute myocardial failure. Recovery was fairly rapid, but we enforced bed rest until dismissal, April 15, 1939.

Subsequent Course.—The patient was recently examined. He is now, eight months after operation, up and about constantly, and supervises a small business. It is astounding that there is not the least swelling or induration at the site of the aneurysm (Fig. 4), nor any thrill or bruit. He has gained 40 pounds. His blood pressure is now 210/120. The Horner syndrome has disappeared. The condition of the right arm is as before operation.

Discussion.—I can find no instance of the treatment of an aneurysm by the method employed in this case. McNealy and Shapiro[1] report the use of pedicled transplants of muscle within the sacs of two aneurysms, one of the third part of the subclavian, and one of the femoral. In both cases, however, the transplants were not inserted until after control of the blood supply by proximal and distal ligation. These writers report some interesting experiments on the effects of loosely inserted pedicled muscle transplants within arteries. They found "that the intraluminal part of the muscle graft atrophied. After one or two weeks it had disappeared, but as it shrunk it was replaced by a severe obliterating endarteritis. No trace of intraluminal muscle or clot was found after three months, but only an intimal cushion which completely, or almost completely, occluded the lumen and left only an occasional narrow, limited channel along the opposite wall. The segment of artery involved was reduced after six months to a fibrotic cord with the muscle graft still attached to its wall."

Reid[2] has done the only experimental work I can find on complete occlusion of arteries with fascial plugs. He states: "When balls of fascia are introduced into the lumen of an artery and anchored by a suture of silk, complete occlusion may be accomplished without much damage to the arterial wall save for some atrophy at the site of the plug. This plug produces complete occlu-

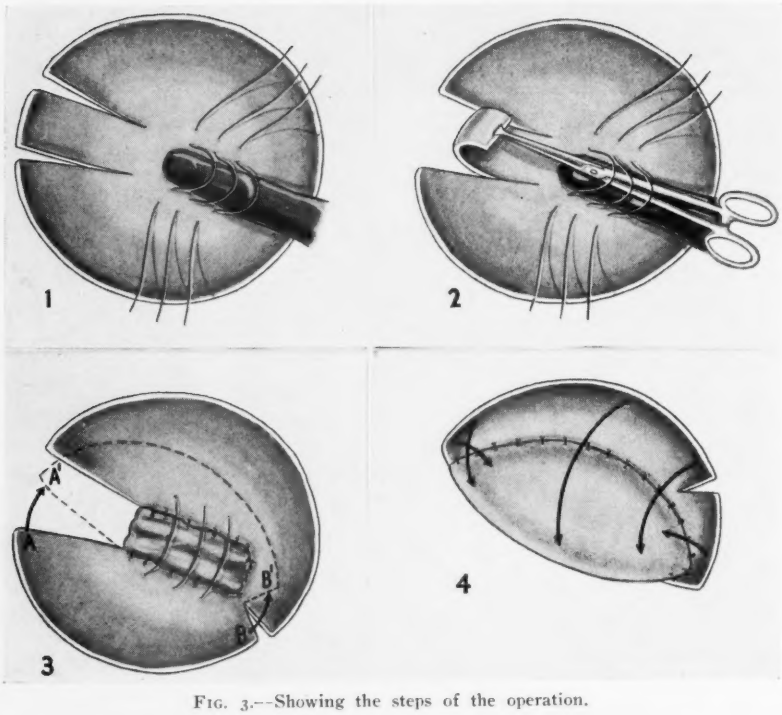

FIG. 3.—Showing the steps of the operation.

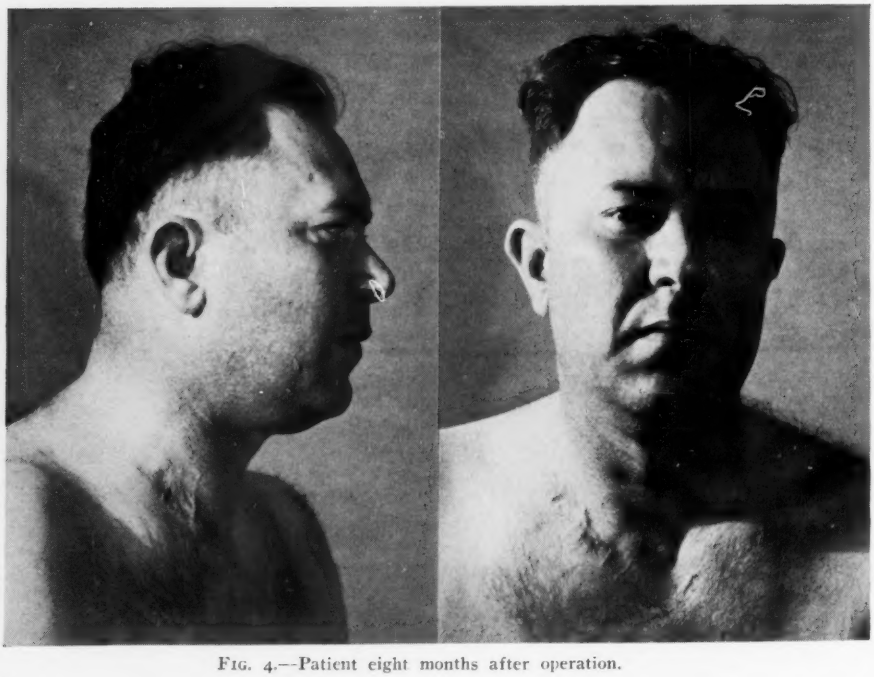

FIG. 4.—Patient eight months after operation.

sion and at its point of contact with the blood stream it becomes covered with intima." He also states that "the fascial plugs remain as viable tissue and do not become substituted by fibrous tissue; that where the fascial plugs come in contact with the blood stream they become covered by intima. For such a method of occlusion there would seem no practical demand." He believes that the organization of thrombi in the region of the plug comes chiefly from the media and adventitia rather than from the intima.

I was greatly impressed by the success, in the case I have reported, of obturation of the artery. A free plug of sac wall would probably have done just as well as a flap. It is unlikely that the distal part of the flap received any nourishment from its own blood vessels. The protocols of the experiments of McNealy and Shapiro convince me that this was true of the circulation in their muscle transplants.

False aneurysm of the subclavian artery is rare because wounds of this vessel are liable to be immediately fatal. Lee, Mitchell and Peacock[3] could find only 129 recorded cases in the 172 years ending in 1934. False aneurysm of the first and second portions of the vessel is extremely rare and its operative cure is regarded by all writers on this subject as always difficult and often impossible. I hope the method I have described may have at least a limited field of usefulness in the cure of these false aneurysms situated above what Halsted has called "the domain of the tourniquet." It involves, of course, free incision of the aneurysmal sac before its blood supply has been controlled, but by use of the plug of sac wall it provides a ready and easily applied method of controlling the hemorrhage. The operation reported could probably have been accomplished without preliminary constriction of the innominate artery, but this certainly added to its safety.

The method would seem well suited for the cure of false aneurysms of long standing. It can be most easily performed when ligation and excision are most difficult. It should certainly not be tried until after the aneurysm has been present for two or three months, that is, until the false sac has become fully developed. This is no disadvantage, however, because it is generally recognized that operation before the formation of the false sac is very dangerous. The method has all the advantages of ordinary endo-aneurysmorrhaphy and gives extraordinarily firm and secure closure of the arterial wound. There is no reason to believe that it is any more likely to cause embolism than is ligation.

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DISCUSSION.—DR. BARNEY BROOKS (Nashville, Tenn.): It would be too bad if the presentation of these two papers were not coupled with at least a recognition of an appreciation of their meaning. I should like first to call attention to the courage shown by Doctor Gatch in undertaking the difficult task of cure of the aneurysm in the patient he presented. Anyone who undertakes the operative treatment of large aneurysms so situated that it is impossible to obtain complete control of the blood supply to the sac, is fully aware of the courage and skill necessary to success from a bold incision into the tense aneurysm sac. James Syme and other pioneer surgeons have graphically described experiences before the days of antiseptic surgery, when this method was the only one which could be employed in the treatment of aneurysm. I assume that Doctor Gatch was unable to expose the common carotid in the neck. This would have given him additional control of the blood supply to the aneurysm sac.

As to Doctor Gage's paper, I would like to point out the fallacy of comparing gangrene after wounds of arteries in war to that following similar wounds in civil life. Certainly, the estimation of the occurrence of gangrene in 40 per cent of cases in which the popliteal artery is occluded is greatly in excess of that we have found to follow obliteration of this vessel in ordinary civil practice. As a matter of fact, we have not encountered a single instance of gangrene following obliteration of the popliteal artery from operations for wounds or aneurysm in this vessel.

I wonder if Doctor Matas considers the case which he presented as perhaps belonging in that group of varying anomalies constituting von Recklinghausen's disease. I remember quite well an operation upon a similar case some years ago in which the patient died from embolism from air sucked into the veins from large blood spaces opened at the time of operation. All of these papers are of extraordinary interest to those of us who have been pupils and followers of Doctor Matas in the development of surgery of the vascular system.

DR. A. W. ALLEN (Boston, Mass.): I also wish to express my appreciation of this symposium. It has been a great pleasure to listen again to the great master, Dr. Rudolph Matas, on this interesting subject. I wish to congratulate Doctor Gatch on the very ingenious manner in which he so successfully carried out the operation upon his patient.

We do not see, in New England, many aneurysms of the luetic variety and have few cases of arteriovenous fistula. However, we have our share of arteriosclerosis and have had a number of instances of sudden thrombosis in the dilated popliteal artery followed by gangrene of the extremity. We have seen a few cases of arteriosclerotic popliteal aneurysm that have come to us prior to thrombosis. In every instance, when these patients have gone on to thrombosis, gangrene has developed. There have been no exceptions in this group. Recently, however, we had a patient with a popliteal aneurysm of the arteriosclerotic variety at a time when Doctor Ochsner was visiting us. He told us of the work Doctor Gage was doing in blocking the sympathetic plexus and advised our trying it. We did so with novocain and then with alcohol, and were much impressed with the diminution in size of the dilated vessel following the procedure. The patient was discharged from the hospital in a few days and, two weeks later, became aware of the fact that the pulsation behind his knee was gone. We were greatly interested to know what was going to happen to him. We expected that he would develop gangrene. He had had a good deal of intermittent claudication. He did not de-

velop gangrene but he continued to have about the same amount of claudication as prior to the thrombosis. I am convinced that the sympathetic block kept him from losing that extremity. For this reason, it is apparent to us that Doctor Gage has made a valuable contribution. He deserves a good deal of credit for calling to our attention the beneficial effect of sympathetic block in such cases.

DR. J. M. MASON (Birmingham, Ala.): In listening to these remarkable and interesting papers, one scarcely knows where to begin a discussion, as there are so many important features connected with each reported case.

In the patient of Doctor Gatch, the important thing is that he was able to control the main arterial blood supply before opening the aneurysmal sac.

I have found that the following steps, by no means original with me, but employed successfully in dealing with aneurysms and arteriovenous aneurysms of the subclavian-axillary vessels, give adequate exposure for any operative attack upon the vessels of this region distal to the anterior scalene muscle:

(1) Skin incision: Beginning at upper border of the thyroid cartilage over the outer border of sternomastoid muscle, down to its clavicular attachment, across the upper surface of the clavicle to its outer third, thence downward and outward to the axillary border.

(2) Dissection and retraction of skin flaps; retraction inward of sternomastoid to expose the scalenus anticus; retraction of this muscle to permit access to subclavian artery as it emerges from behind the muscle.

(3) Division of the clavicle; division of the clavicular attachment of the pectoralis major, of the subclavius muscle, and of the tendon of the pectoralis minor.

(4) Retraction of the divided clavicle to permit access to the subclavian vein as it crosses in front of the scalenus anticus and passes beneath the clavicle.

(5) Reconstruction of the wound in proper anatomic planes, after the vascular lesion has been dealt with.

It may be well to call attention to certain points concerning operations upon vascular injuries or disease in this region.

The subclavian vein should be occluded proximally before any opening is made into this vessel. Failure to do this has resulted in fatal air embolism, as reported in Makin's series.

The subclavian artery should be occluded proximally before freeing the clavicle from an aneurysmal tumor. The clavicle has sometimes been found so eroded that it forms part of the sac wall, and in separating it, the aneurysm may be opened with disastrous results.

The treatment of the divided clavicle has been the subject of some discussion. It is claimed that no material disability follows its removal, and in the one case in my series where the anterior half was removed, the function of the shoulder and upper extremity was not impaired. In reviewing a series of 19 cases, the disposition of the clavicle was not noted in three instances; it was wired three times, and removed, wholly or in part, in eight instances. I have removed the inner half in one instance, and have wired the divided bone in three cases.

Undoubtedly, the presence of a compound fracture of the clavicle protracts the convalescence, and, in the case of infection, the period of recovery may be much delayed.

DR. JOSEPH A. DANNA (New Orleans, La.): We sometimes make an incision into an aneurysm without knowing we are dealing with one. In such

an event, if you put your finger in the incision you have made, at the same time that you control the external flow of blood, you can feel the blood spurting in through the main opening and circulating in the sac. By applying the finger to the easily palpated aneurysmal opening, the blood flow is stopped and the aneurysm may be ripped open and its openings sutured. I have done this on two occasions. In some cases there may be a second or third vessel opening into the cavity. In that case put your finger in one, have your assistant put his finger in the other, and you can go on with your work.

DR. J. L. CAMPBELL (Atlanta, Ga.): Vascular surgery is a field in which we owe much to Doctor Matas. He has been the real pioneer and his work has inspired all of us who have come in contact with patients having lesions of the blood vessels.

Prior to 1930, when I had charge of the Surgical Department of the Emory University division of Grady Hospital, I saw many interesting aneurysms. The most difficult ones to handle are those of the subclavian and the femoral in Scarpa's triangle.

Doctor Gatch is to be congratulated on the successful outcome of his case. If we remove the whole clavicle, or at least the inner two-thirds of it, the approach to the vessels is much easier. Then we are able to expose them more easily, both proximal and distal to the anastomosis. A Dakin tube can be passed around them and clamped without danger of injury. While this will not always insure a dry field, it will enable us to determine whether we wish to attempt a restoration or simply perform a quadruple ligation and resection. Quadruple ligation without resection will not insure a cure. Resection of the vein and artery between quadruple ligatures rarely causes any trouble, for the collateral circulation is almost invariably well established. When the clavicle has been resected subperiosteally it will cause no inconvenience to the patient. One of my patients drove a dray ten years after his clavicle was removed. I doubt if he ever missed it or he may never have known that it had been removed. I have resected or removed the clavicle in ten or more patients without any functional inconvenience.

We should always try to restore the continuity of the vessel in fistulae of the common carotids and internal jugular vein. Of course, the collateral circulation can be stimulated by daily pressure on the vessels at the point of anastomosis and, when all indications of cerebral anemia have disappeared, the vessels can be resected; but when the lumen of both has been restored you can feel much more comfortable. This is the most favorable location for a restorative procedure because there are no branches to be encountered or controlled; simply expose the artery and vein above and below the fistula, pass the Dakin tubes around them and clamp. Then separate the vessels and suture the opening. In my early cases I placed a strip of muscle over the suture line, then over that a band of fascia. In my later cases I only placed a strip of muscle between the vessels and closed the sheaths.

Repair of an arteriovenous fistula in Scarpa's triangle gives more trouble than in any other location. In three cases I felt sure the fistula was between the femoral vein and artery, but after a further search I found it about 1 cm. from the femoral in the profunda. There is no debate in one's mind about the procedure to take in these; for it is easy to perform a quadruple ligation and divide the vessels at the fistula. If the vessels are not divided and the sac removed (if there is one), the patient will not be cured.

DR. MIMS GAGE (New Orleans, La., in closing): I wish to thank both Doctor Brooks and Doctor Allen for their kind discussion. I am very glad

that Doctor Allen has had success in developing the collateral circulation in his cases of peripheral aneurysms by blocking the sympathetics. I think that Doctor Brooks misunderstood my remarks in regard to the high incidence of ischemic gangrene in war wounds of the femoral and popliteal arteries. It was my purpose to demonstrate that the inherent qualities of all aneurysms are to develop collateral circulation, whereas the sudden occlusion of a normal peripheral artery invokes a high incidence of ischemic necrosis. I think that Tuffier reported an incidence of 66 per cent gangrene in 24 ligations of the popliteal artery in contrast to Matas' 5.2 per cent of gangrene following 154 personal operations for popliteal aneurysms, clearly demonstrating the differences that exist between normal peripheral vessels and those diseased with aneurysms. Another typical example is the high incidence of gangrene that occurs following peripheral arterial embolism. I believe both the above comparative pictures definitely indicate that release of vasospasm of both the main arterial tree as well as the collaterals is of foremost importance before ligation of the peripheral arteries in the treatment of aneurysms is undertaken.

Dr. Rudolph Matas (New Orleans, La.): There is such a wealth of information in Doctor Gage's and Doctor Gatch's papers and so much that is new and informative in the discussions that followed, that it seems to me we could spend the entire evening profitably by going over the ground they have covered. I feel that the Chairman has been so indulgent and the members who have spoken have been so generous in their praise of my unpretentious work that it would be best that I should only thank them—which I do very sincerely—and say no more. I would, however, like to avail myself of this opportunity to express my heartfelt gratitude to many of my former pupils, here and elsewhere, whose loyalty and generous praise touch me deeply. After hearing Doctor Gage's frequent and flattering references to my vascular hobbies, you should not be surprised if I blew up with vanity.

Doctor Gatch's operation was such a brilliant performance that it deserves our warmest congratulations. Those who are not immersed in this kind of surgery can scarcely realize the great tension, the anxious moments, and the worries that are involved in these deep-neck performances. Doctor Gatch's operation is, I believe, unique in utilizing a flap from the sac to close the chief orifice of arterial supply. The whole conduct of the case shows great ability to think quickly and act successfully, on the spot, in a critical situation.

Again, gentlemen, I thank you all for your kindness to me!

HYSTERICAL EDEMA OF THE HAND AND FOREARM *

CARRINGTON WILLIAMS, M.D.
RICHMOND, VA.

MANIFESTATIONS of major hysteria which simulate surgical lesions are numerous and well known. Such conditions are not usually accompanied by change in the structure of the part involved but rather are manifested by an apparent loss of function. Hysterical seizures are usually defense reactions and the major examples are designed by the individual as a means of protection against problems of life which he feels unable to successfully combat.

Edema in localized areas of the body may be the result of a number of factors. Among these is venous congestion (Loeb[1]). When such congestion is due to increased venous pressure, there is dilatation of venules and capillaries from this increased pressure and an increased exudation of fluid into the tissue spaces. As fluid increases in the tissues, the pressure here is increased and there are also changes in the osmotic tensions of both inorganic salts and proteins, so that a point may be reached where exudation will cease when the tissue pressure equals or exceeds the combination of osmotic and vessel pressure (Youmans *et al.*[2]).

When there is interference of outflow of fluid from the tissues through both the venous and lymphatic channels, this increased tissue pressure results. Youmans[3] has shown that there is a marked increase in the circulating time of the blood in the leg on quiet standing. He has also demonstrated a considerable increase in leg volume after quiet standing. The speed of blood flow is increased by motion and increase in volume does not occur. He believes that much more fluid is actually passed from the capillaries into the tissue spaces during this period, but it is removed by the improved venous and lymphatic return circulation. The amount of blood passing through the small vessels is also increased by the improved return and this promotes increased exudation of fluid.

Wells, Youmans and Miller[4] have found increased tissue pressure in various parts of the leg after periods of quiet standing and have found that marked increases occur in muscles which are covered by stout fascia, such as the anterior tibial group, a much smaller increase is present in the gastrocnemius which has a thin fascia, and only a slight increase is found in the subcutaneous tissue. Landis and Gibbon, Jr.,[5] demonstrated edema in the forearm from congestion produced by a pneumatic cuff on the arm and measured by a "pressure plethysmograph" on the forearm. This apparatus eliminated measurement of the increase in forearm volume due to increased blood in the veins. During the first part of the experiment the venous congestion accounted for the major portion of the increased volume, but after

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30 minutes the congestion did not increase, so that later increase in volume was due to increased fluid in the tissues. They found that the filtration rate increased with increasing venous pressure, and with a constant venous pressure the rate declined with increase of tissue pressure.

Many cases of hysterical paralysis have been reported but in most of these cases the lower extremities are involved following an injury to the back. Parker[6] reported a case of hysterical paralysis of the arm in a young

FIG. 1.—Case 1: Condition of arm on admission to hospital.

woman. There was no circulatory disturbance in this case because the position of the forearm was either held at a right angle to the arm by bandage or the position was frequently changed.

A search of the literature (admittedly not complete) of the last 20 years fails to find a report of edema of the forearm and hand produced by the immobile dependent position of the extremity as a result of hysteria or malingering. The three cases reported herewith demonstrate this condition and the three experiments confirm the clinical impressions.

CASE REPORTS

Case 1.—A. T., female, age 14, was admitted to Sheltering Arms Hospital, May 2, 1931, complaining of painful swelling of the left hand and forearm. She stated that eight months before she had cut a finger on a ring and that the swelling began around this laceration and extended over the hand and forearm (Fig. 1). She had been under the care of a number of physicians; the treatment had been varied and included three incisions into the hand. None of these openings discharged pus. The swelling has continued without relief. She claims complete inability to move the hand and wrist. Her past personal history was unessential.

Physical Examination.—The patient was well developed and well nourished. No abnormality was found except the swelling of the left hand and forearm, which were greatly increased in size, pitted on pressure, and the skin, particularly on the dorsum of the hand, appeared thin, red, and hot. The picture suggested acute inflammation. The blood count and uranalysis were normal. Roentgenologic examination of the bones of the hand and forearm failed to reveal any lesion.

Incisions of the hand was being seriously considered when one of my colleagues (Dr. G. Paul LaRoque) suggested that the edema might result from the position and immobility of the part. In order to test this theory the patient was kept in bed with the hand elevated and fixed. In about 12 hours the edema had completely subsided and the hand appeared normal. She was then allowed to be up and in a short while the whole picture recurred. It was observed that she held the arm immobile in a dependent position and at night held it downward over the side of the bed.

A more careful search was then made into her family history and it was ascertained that her mother had died when she was a small child. Her father had married again and there were several children by this second marriage. Her stepmother had always been kind and affectionate to the point of greater indulgence than to her own children, but this child had not been happy. Psychiatric consultants were positive in the diagnosis of major hysteria. She was kept under observation and treatment in the hospital for a number of weeks until she was entirely cured. Except for a few minor recurrences she has had no further trouble. She is now married, has children of her own, and is leading a normal life.

Case 2.—C. F., white, male, age 40, was admitted to the Church Home and Infirmary, Baltimore, Md., on the service of Dr. William F. Rienhoff, Jr., April 15, 1935.

The left hand and forearm were greatly swollen, red, and tender (Fig. 2). This condition had existed during the previous 18 months. He had had a similar illness several years previously, until he was cured by a medicine man of the Hopi Indians in Arizona. During the preceding few months he had been studied in clinics in other cities where a variety of diagnoses were made and a number of surgical procedures advised, including amputation of the arm. There was a history of an excellent background for hysteria. The arm and forearm were first held elevated in a plaster encasement and later by suspension. The edema gradually subsided entirely but the small joints of the fingers were permanently damaged by the long-standing immobility (Fig. 3). He was kept in the hospital for a number of weeks and was finally persuaded that his disability had been entirely functional. At present there is no edema, but the mobility of the fingers has not been restored (Fig. 5).

Case 3.—This case is reported because it so closely resembles the others, but it has not been so adequately proved. The patient, an elderly Chinese beggar, was admitted to the Ellen Lavine Graham Hospital, in China, on the service of Dr. John H. Reed, Jr., complaining of swelling of the left hand and forearm which he stated followed a minor scratch of the skin many months previously.

FIG. 2.—Case 2: Condition of arm on admission to hospital.

FIG. 3.—Case 2: Condition of hand six weeks after admission to hospital.

The swelling increased the size of the parts to at least twice that of the opposite side (Fig. 4). There were several clean, shallow areas of ulceration on the hand, but no evidence of acute infection. Several linear incisions were made over the forearm and fingers, from which a thin, clear fluid was discharged. After a short period in the hospital, the swelling almost completely subsided; there was no discharge of pus.

FIG. 4.—Case 3: Condition of arm on admission to hospital.

SUMMARY.—Two of these individuals were of the "neurotic" type with good backgrounds for hysterical manifestations. The third, a beggar, was a malingerer who used the self-produced deformity as an asset in his trade. It is interesting to note that the left hand was involved in all three. Their ages differed widely; two were males and one a female. All recovered without residual swelling, but the small joints of the hand in Case 2 were so damaged by the long-standing immobility that their function has not been restored.

EXPERIMENTAL DATA:—In order to measure the changes in the normal forearm and hand resulting from the immobile, dependent position, the following observations were made on each of three subjects. Measurements of diameters at fixed levels were made with calipers. Measurements of circumferences were made with a tape. Measurements of volumes of the hand, lower forearm, and upper forearm were made by immersing the part, to the same levels, at intervals of one-half hour, in a cylinder of water and determining the amount

of water displaced. The results of these measurements are shown in Tables I and II.

TABLE I

CHANGES IN MEASUREMENTS OF THE HAND AND FOREARM RESULTING FROM DEPENDENT, IMMOBILE POSITION FOR FOUR AND ONE-HALF HOURS

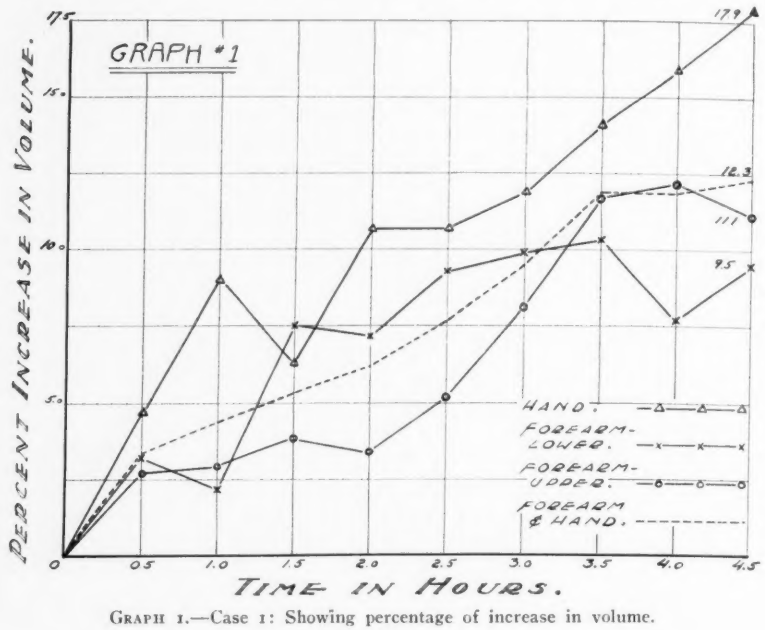
	Second Finger		Palm		Forearm	
	Diameter in Mm.	Circumference in Mm.	Diameter in Mm.	Circumference in Mm.	Diameter in Mm.	Circumference in Mm.
Case 2						
Beginning	19.0	62.5	30.0		65.0	244
End	22.0	71.0	36.5		73.5	247
Case 3						
Beginning	20.8	68.0	28.5		64.0	230
End	24.0	75.0	34.8		73.0	242

TABLE II

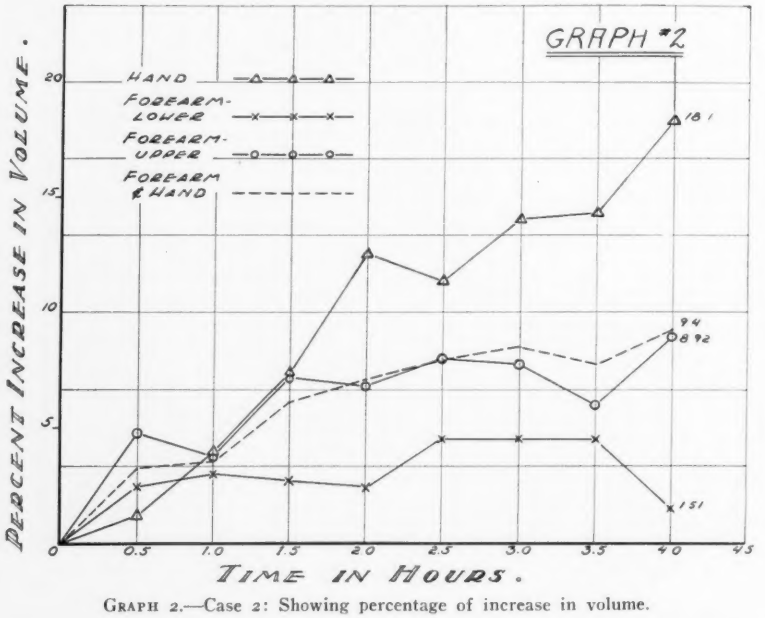
CHANGES IN VOLUME

	Volume of Hand	Increase in Volume	Volume of Lower Forearm	Increase in Volume	Volume of Upper Forearm	Increase in Volume	Total Volume of Hand and Forearm	Increase in Volume
	Cc.	Per Cent	Cc.	Per Cent	Cc.	Per Cent	Cc.	Per Cent
Case 1								
Beginning	447		494		928		1,869	
End	527		541		1,031		2,099	
Increase in volume	80	17.9	47	9.5	103	11.1	230	12.3
Case 2								
Beginning	328		332		650		1,310	
End	388		337		708		1,433	
Increase in volume	60	18.1	5	1.5	58	8.9	123	9.4
Case 3								
Beginning	357		330		655		1,342	
End	425		323		719		1,467	
Increase in volume	68	19.0	−7	−2.1	64	9.8	125	9.3

A graphic presentation of the percentage of the increases in volume in Cases 1, 2 and 3 is shown in Graphs 1, 2 and 3.



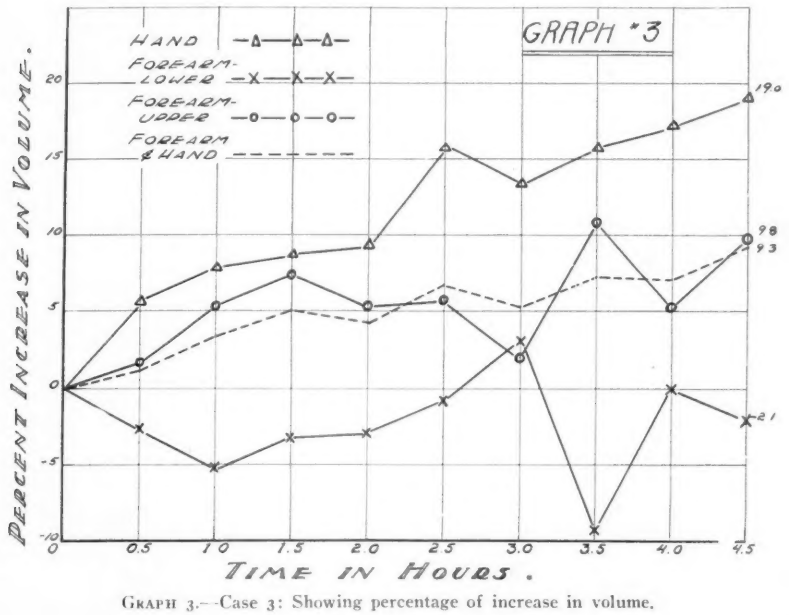
GRAPH 1.—Case 1: Showing percentage of increase in volume.



GRAPH 2.—Case 2: Showing percentage of increase in volume.

The results were uniform for the three subjects except the volume of the lower forearm which showed a substantial increase in volume (9.5 per cent)

in the first, a slight increase (1.5 per cent) in the second, and a decrease in the third (−2.1 per cent). All of the other measurements showed a steady increase in volume.



GRAPH 3.—Case 3: Showing percentage of increase in volume.

FIG. 5.—Experiment 2: Shows the appearance of the normal left arm after being four hours in a dependent immobile position.

At the end of the first hour, a distinct enlargement of the dependent hand could be observed, and at the conclusion of the experiment it was quite marked (Fig. 5). After concluding the experiments, each subject complained of some pain, numbness, and stiffness of the hand; but after using it normally for an hour, the edema subsided and normal sensation and mobility were restored.

CONCLUSION

These three cases illustrate a unique manifestation of major hysteria which simulates surgical lesions. That the edema resulted from the dependent, immobile posture of the arm was demonstrated by the prompt recovery of these patients by change of position with the addition of minor incisions in one. The experiments further verify this conclusion.

I am indebted to Doctors Reed and Rienhoff for permission to report their cases, and to Dr. John L. Patterson, Jr., and Carrington Williams, Jr., for performing the experiments.

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CAISSON DISEASE WITH SPECIAL REFERENCE TO THE BONES AND JOINTS*

REPORT OF TWO CASES

BRADLEY L. COLEY, M.D.

AND

MOORE MOORE, JR., M.D.

NEW YORK, N. Y.

AMONG the unusual nonmalignant osseous lesions sometimes encountered are those produced by caisson disease. This condition has received but scant attention in medical literature. Not infrequently it has been discovered by accident in the course of radiographic examinations conducted for other purposes.

It seems worth while to the authors to point out the characteristic clinical and radiographic features of this process, and to emphasize the fact that it must be regarded as one of the causes of late arthritic changes which have an occupational basis.

Aristotle mentions a metallic vessel containing air when lowered into water, and records that the diving helmet was used as part of the equipment of Alexander the Great. At the Siege of Tyre (333 B.C.) divers removed obstructions and loosened anchors and cables. Julius Caesar describes a varnished leather apparatus for diving and both Roman and Greek navies employed divers.

Although Triger, a French engineer, in 1839, was the first to successfully operate upon a case of caisson disease, men have been working in various capacities under increased air pressures either in caissons or diving suits since the sixteenth century. He, incidentally, was the first to note and emphasize the extremity pains which cause subjects to assume the characteristic attitude known as "bends." However, Paul Bert,[4] in 1871, was the first to describe such cases clinically and to advance the correct theory of their etiology.

Though this paper deals chiefly with the clinical and radiographic features of skeletal manifestations, a brief résumé of lesions by systems is included:

(1) Cerebrospinal: The symptoms range from numbness and tingling to unconsciousness and collapse. The special senses are frequently involved, Ménière's symptom-complex being noted often.

(2) Cardiovascular: Manifested by embolism.

(3) Pulmonary involvement is shown by dyspnea (1.5 per cent of Erdman's cases). This is always a grave symptom for it is evidence of massive embolism, multiple in the lungs.

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(4) Visceral and Urogenital: Symptoms are rare; nausea, vomiting and epigastric pain, however, also being grave signs indicative of massive embolism.

(5) Skeletal System (osseous, connective, muscular and fatty tissues): Pain is the dominating symptom, occurring either alone or with other symptoms in from 85 to 90 per cent of the cases. The myalgias, ostalgias and arthralgias are described as "tearing, boring, gnawing or lancinating." Mild attacks are felt by almost all workers.

(6) Dermal: Pruritis is very common and may be the first and only symptom. Lividity is associated with serious cases.

No actual figures as to the relative incidence of lesions according to systems are found except by Quadri,[17] who states that the following, in decreasing order of frequency, has been his experience: (1) Muscular and articular forms (of sudden onset with rapid course and favorable prognosis). (2) Auricular: The lesions being in the internal ear. (3) Central nervous system: With lesions affecting locomotion. To these cases of sudden onset he also gives a good prognosis. All writers agree that dermal symptoms are most common for the incidence of the disease is almost 100 per cent, if trivial cases are counted.

It is well established that all symptoms and lesions are the result of actual bubble formation in the circulating blood or in various tissues. These are caused by the inability of the system to excrete rapidly enough via the lungs, the excess nitrogen which has been held in supersaturation in the various tissues under increased air pressure.

The blood is almost instantly saturated at the partial pressure of gas in the lungs. The kidneys (Hill and Greenwood[10]) saturate and desaturate ten times as rapidly as the body, as a whole, while the body becomes completely saturated in five hours (Haldane).

The absorptive power for nitrogen of fats and lipoids is five to six times greater than that of blood, and they, along with water, are the chief solvents of nitrogen. During the process of saturation, a large part of the nitrogen, absorbed by fat and lipoids, diffuses to them from body fluids. On decompression the reverse is thought to occur.

The tissues with poorest blood supply, in relation to nitrogen content, are the bone marrow and spinal cord. These tissues with high nitrogen content are enclosed in bone, thus preventing free diffusion of nitrogen into contiguous tissues and so making them dependent upon a comparatively poor blood supply for absorption and elimination. Consequently there is a retardation of nitrogen elimination from these tissues when bubbles are present in the blood, and when the pressure-head of nitrogen in the tissues is high such delay is apt to be prolonged.

In 1908, Boycott, Haldane and Damant[6] showed that the only extravascular bubbles were those found between the fat cells of the cord and other areas rich in adipose tissue. No bubbles were ever noted in the cells them-

	Per Cent of Fat	Per Cent of H_2O	N_2 Content (Cc./100 Gm.)
Body as a whole.....	15.4	59.0	1.4
Brain.....	4.8	70.0[1]	0.94
Spinal cord.....	27.8	70.0[1]	2.2
Bone marrow.....	90.0		

[1] Estimated = 1.

selves. It has been shown that nitrogen bubbles grow by accretion, and pressure in excess of the working level has been used to reduce the size of the bubble in recompression therapy, which accomplishes little in the way of removal since on rapid decompression the symptoms returned. In this instance helium may be used to displace nitrogen since it is inactive and has none of the toxic properties of oxygen. Naturally, the treatment is largely preventive, by slow decompression. Specific therapy of bones and joints will be discussed later.

Location of Bone and Joint Lesions and Their Relationship to Each Other.—The long bones are the favored areas for both symptoms and lesions; possibly the vertebrae and membranous bones escape, due to the high proportion of red as compared to fatty marrow. Erdman[8] reports about 70 per cent of symptoms in the lower extremities, chiefly the knees; the remainder being in the upper extremities. These figures agree with those of other writers. Also, he aspirated under water a few cubic centimeters of gas from under the periosteum of the tibia of two workers.

The location and severity of symptoms depend upon the site and volume of gas set free. Pain results when it is confined beneath any unyielding tissue (muscle spindles, ligaments, fascia, periosteum and nerve sheaths). Symptoms are rare in the liver, spleen, kidney, fat deposits or veins, and these latter "silent" areas outnumber the "painful" ones which in turn outnumber the "vital" ones, *i.e.*, the central nervous system. Symptoms have occurred during decompression, but are most common during the first few minutes after decompression. Erdman, in 3,692 cases, noted 50 per cent within 30 minutes, 95 per cent in three hours, but 1 per cent delayed over six hours. Four cases occurred between 15 and 23 hours. Reexposure after incomplete decompression is known to predispose to attacks, as is the use of alcohol shortly prior to exposure. Infarction from an old injury may determine the site at which a lesion locates.

Location of Lesions.—German authors describe 11 cases, all of which present lesions involving a large joint. The hip is affected 12 times, the shoulder once. All cases, with two exceptions, are monarticular; these are both bilateral, in the hips. They all agree with Phemister, Kahlstrom and Burton[13] that the pathology is a result of nutritional interference either from direct embolism in a main nutrient vessel or of pressure on the vessel wall by bubbles, or both. This sets up a train of infarction plus aseptic necrosis of

varying extent. Following this is the classic picture of gradual attempts at repair on the part of the bone by osteoclasis, creeping substitution and eventual delimitation by calcification as described elsewhere.[13] Also, these German authors are of the opinion that the characteristic picture of alternating areas of translucency and density, with a secondary arthritis, is not the result of one exposure, but rather of repeated insults. All agree that the arthritis, which is of the deforming type, is a result of absorption, collapse and new bone formation in, or near, an epiphysis. Only one pathologic specimen was examined by them, and that only in the gross; the head of the femur and capsule being described as having many knobby excrescences. The German authors make no mention of extensive areas of aseptic necrosis extending far along the shafts of the long bones from the initial diaphyseal lesion. The vessels in this region have few anastomoses and are, therefore, easily clogged, which fact, probably, influences the location of the lesion.

Twynam,[25] in 1888, reported a case of a man who had severe symptoms of caisson disease with pain and swelling above the right knee. Two months after the onset an abscess was drained there and later another one near the right major trochanter. Due to persistent sinuses, amputation, through the lower third of the femur, was performed two years later. The entire shaft of the femur was found to be necrotic and surrounded by an involucrum one-third of an inch thick. This seems to have been a case in which massive necrosis of the femoral shaft plus secondary infection produced a picture simulating pyogenic osteomyelitis.

Kahlstrom, Burton and Phemister[13] report four cases of long standing, one of which was proved by autopsy and one by biopsy. All were studied clinically and roentgenographically.

Molfino[15] states that 27 of 30 cases had joint pains, mainly in the lower limbs and especially in the knees. He quotes the following descending order of frequency: Knee; shoulder; elbow; and hip. Motion, both active and passive, he states is not painful and articular pains persisted for only a few days. The long bones, especially the tibia, were found to be tender in the acute stage. Roentgenograms taken three to four days after onset showed no noteworthy changes.

Bassoe,[1] in 1913, reports 15 cases of which nine can be said to have definite bone or joint pathology demonstrable roentgenologically or clinically. The lesions were located as follows: Hip 6; knee 4; tibia 2; fibula 1; shoulder (humeral head) 1; ankle 1; talus 1. It is noteworthy that one case which had arthritis in both ankles and the right knee had only the one exposure after working under a maximum pressure of 26 pounds. The talar lesion showed a mushrooming of this bone into the navicular with stiffening of the ankle, as well as islands of sclerosis and atrophy in the tibia, but also had evidence of residual cord disease, thus giving rise to the question of trophic osteo-arthropathy.

No authors report pathologic fracture, except perhaps Bassoe, who de-

scribes a case which had a spontaneous patellar fracture 11 years after "the bends." Roentgenograms showed osteoporosis and atrophy of the fragments at the time, but healing was not unusual. This patient also had permanent cord damage due to the disease.

The specific therapy of bone and joint lesions is, naturally, that of prevention and correction of deformity from contracture at the affected joints. In the event of severe deforming arthritis, arthrodesis and arthroplasty must each be considered in relation to the individual case.

The importance of recognition of both clinical and roentgenologic features of this disease, with its intimate relationship to possible compensation claims, is self-evident. There is one case recorded of arthritis deformans of the hip as an alleged result of caisson disease, in which causal relationship was not established and compensation was denied (Jaeger[12]). This, aside from purely legal aspects, apparently was adjudged chiefly on the grounds that it was an occupational disease and did not occur as the result of one attack. Also, the first signs were noted three years after the exposure and came on intermittently. No roentgenograms of the long bones were published and they were thought to be consistent with early deforming arthritis, although one expert considered it to be caisson disease.

Roentgenographically, caisson disease may be distinguished by multiple distribution in the medullary portion of the bone, rarely, if ever, producing cortical change. The shaft is not expanded and the infarction presents as an area of irregularly increased density usually surrounded by a thin band of calcification on the outside of which is a variable amount of normal appearing medulla. The lesion is located in the diaphysis and may, or may not, involve the epiphysis and secondarily the joint. Articular changes resemble arthritis deformans. The lesions almost always are located in the large long bones, namely, the femur, tibia and humerus and more often in the lower than in the upper extremity.

The diseases most likely to be confused in diagnosis are: (1) Chronic sclerosing osteitis which is not usually multiple and in which there is evidence of cortical thickening and narrowing of the medullary cavity. (2) Low grade sclerosing osteogenic sarcoma in which there is definite pain far in excess of that in caisson disease, and the area of involvement fades by insensible gradations to normal tissue; also, there is no sharply delimited calcific boundary. (3) Calcifying enchondroma which, although frequently multiple and symmetric, is usually expansile and does not involve the joints. (4) Lues can be differentiated by periosteal reaction and serologic tests. (5) Tuberculous diaphysitis usually shows laminated new bone along the shaft and does not have the irregular outline plus the peripheral band of calcification; it also is not multiple, and supporting stigmata may be found elsewhere in the body. A history of exposure to compressed air can be elicited always, and this is the deciding point.

The roentgenographic picture is easily explained by the pathology. This has been shown[13] to consist of a central area of dead bone whose irregular outline is due to partial replacement by new bone and new marrow. When the replacement process ceases the region is bounded by a band of calcification. Small areas can be replaced entirely by dense new bone. Joint changes are dependent upon the same process plus the added factor of weight bearing in many instances.

Thus the entire picture may be explained on the basis of vascular insufficiency and nutritional disturbance in their late stages.

CASE REPORTS

Case 1.—M. H., male, white, age 57, was a compressed air worker who was first treated at the Memorial Hospital, July, 1938, for a squamous carcinoma of the tongue. The patient had worked as a "sand hog" for about 16 years, during which time he had many trivial and at least eight definitely recognizable attacks of the "bends." The first attack was about 1904; the last in 1918. In November, 1914, while working on the Colorado River Irrigation project, at 38 pounds' pressure and in intense heat (to about 130° F.), the patient suffered his most severe attack immediately after coming up from the decompression terminal. At this time workers alternated one hour of rest after each hour of labor for a total of eight hours. The patient suffered collapse and unconsciousness, which were of short duration. He experienced aphasia for two days and loss of urinary sphincter control for three to four days, but no loss of rectal sphincter control. No visual or auditory symptoms were noted, but he did have a complete right hemiplegia, lasting 13 to 14 days. His recovery was rapid and he returned to work three weeks after the incident, able to do a full day's work. During his convalescence he had marked muscle and joint pains referable to the right upper and lower extremities.

He also had an attack in 1918 in New York Bay, at which time the abdominal wall became discolored but there were no other remarkable symptoms. In all attacks pain was referable especially to the right hip and shoulder. There was never any joint swelling or discoloration. After the 1914 incident, patient noted a gradual stiffness of the right hip and shoulder, being definitely noticeable in a few months' time. He had pain, of a dull nature, which was somewhat aggravated by raw weather, though it had been less in the past two to three years. The left knee had had milder similar pains.

Physical Examination.—The right hip is almost completely ankylosed in about 10° flexion and 10° adduction.

Measurements:	Right	Left
Ant. sup. spine to internal malleolus	86.0 cm.	92.5 cm.
Tibial tubercle to internal malleolus	34.5 cm.	34.5 cm.
Thigh circumference (20 cm. above tibial tubercle)	37.5 cm.	40.5 cm.

The patient stands with a marked elevation of the right pelvis and a compensatory lumbosacral scoliosis. Patient walks with a definite right hip limp.

Right upper extremity: External rotation is *nil*. Internal rotation about one-half normal. Glenohumeral abduction to 35°; combined with scapular motion and anterior elevation to 75°. Patient can just put his hand on the iliac crest and comb his hair. There is some crepitus in the shoulder with motion, but no pain.

Neurologic Findings:	Right	Left
Knee jerks	Two plus	Three plus
Ankle jerks	One plus	Two plus
Biceps (humeri)	One plus	Two plus

FIG. 1.—Case 1: Showing a characteristic medullary infarct in the right femur.

FIG. 2.—Case 1: Showing the tibial lesion.

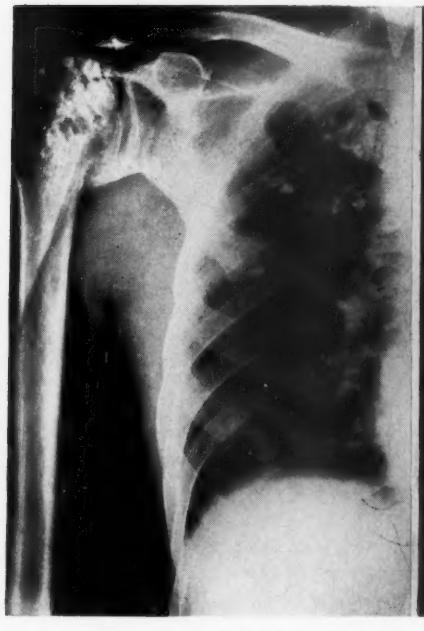

FIG. 3.—Case 1: Showing the arthritis deformans—note the osteophytes.

FIG. 4.—Case 1: Showing the arthritis deformans of the right hip with almost complete fusion.

There is a left Horner's syndrome.

Roentgenographically, the patient presented lesions in the following areas:

(1) Right lower femoral diaphysis (Fig. 1).
(2) Right upper tibial diaphysis (Fig. 2).
(3) Right shoulder and humeral head (Fig. 3).
(4) Right hip and femoral head (Fig. 4).
(5) Left lower femoral shaft.
(6) Left humeral head (small).

Other than the right hip and shoulder, the lesions were clinically "silent."

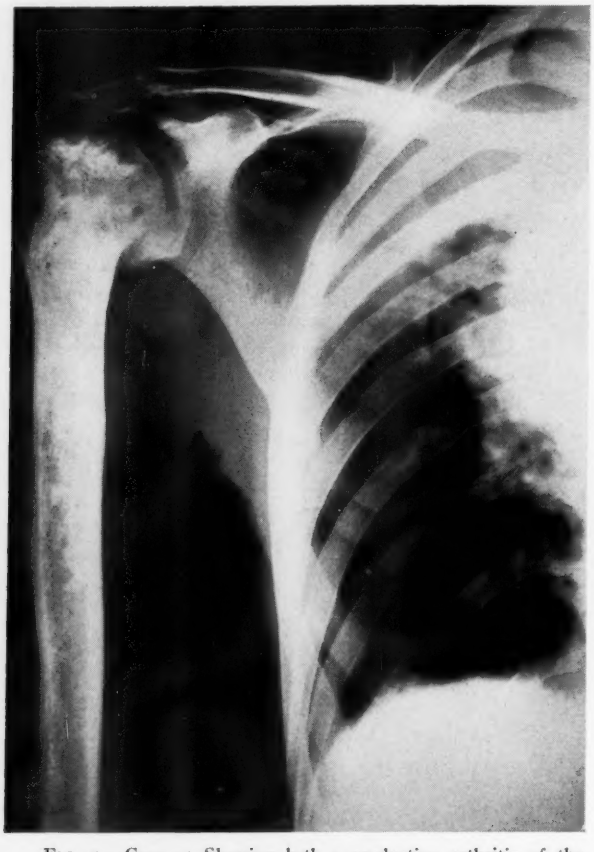

FIG. 5.—Case 2: Showing both a productive arthritis of the right shoulder joint and extensive diaphyseal lesions of the humerus.

Case 2.—W. R., Negro, male, age 49, was first seen in the Memorial Hospital, December, 1938, at which time he was referred to us for treatment of bronchiogenic carcinoma, arising from the right main bronchus. A routine roentgenogram of the chest included a portion of the right humerus, which showed a huge infarct in the shaft of the bone, and views of the humerus itself were then obtained, but at that time we were unable to diagnose the lesion. On being questioned, at a later date, the patient gave the following history: In 1930, patient worked as a "sand hog" on the Erie Tunnel project, working in pressures up to 40 pounds. He was thus employed for about four months, and this was his only connection with compressed air work. About two to three days after commencing it he had had a very trivial attack, noting some soreness and mild pain with slight subsequent stiffness referred solely to the shoulders and arms. About two

weeks later he came up from the decompression terminal, after working at 30 pounds' pressure, complaining of severe pains, particularly in his shoulders and both entire upper extremities. There was pain in the lower extremities to a lesser degree, chiefly the knees. He did not lose consciousness or become paralyzed. Within 20 minutes, after the onset, he was replaced in compressed air and the pain was slowly alleviated, requiring about eight hours. There were no central nervous system symptoms whatsoever. Following this attack there was extensive stiffness and soreness of both upper and lower extremities, the former locale being more noticeable. However, he continued to work under varying pressures with a steady diminution of symptoms; all disappearing in two to three weeks. He then noted a gradual loss of the normal mobility of right shoulder, but had no joint pains there. The patient suffered no other attacks of the "bends." In January, 1939, he experienced a moderately acute attack of arthritis in his left wrist. Again, in May, 1939, he had a similar episode affecting the left shoulder. Both responded to symptomatic treatment and in neither was an etiologic agent determined, or significant roentgenologic changes noted.

Physical Examination.—Ankles, knees and hips normal. Right shoulder: Atrophy of soft parts of the girdle. Internal rotation about 10°–15°. External rotation 0°–5°. Glenohumeral abduction to 40°; combined with scapular motion and anterior elevation this increases to 75°. He can just put his hand on the iliac crest. Right elbow and wrist negative. No reflex changes noted.

Roentgenographically, this patient showed lesions in the following regions:

(1) Right humeral shaft and shoulder (Fig. 5).
(2) Left lower femoral shaft.
(3) Left upper humeral shaft (small).
(4) Left upper tibial diaphysis (small).
(5) Right upper tibial diaphysis.
(6) Both ischii (small).

The lesion in the right shoulder was the only one giving clinical signs.

SUMMARY

The available literature referable to caisson disease, particularly as exhibited in the skeletal system, has been reviewed.

It seems quite likely that the characteristic lesions can result from one insult of sufficient severity.

The late effects of nitrogen gas bubble formation in the tissues and circulation form a recognizable picture, both clinically and roentgenographically, the importance of which, in the field of compensation claims, has been pointed out.

Two cases have been presented. Each of these has lesions which are clinically silent in addition to the evident arthritis.

Attention is called to caisson disease as a possible cause of vague bone and joint pains which may be discovered later as "silent" areas of aseptic necrosis.

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DISCUSSION.—DR. ISIDORE COHN (New Orleans, La.): While I have seen none of these cases, we can never tell when some of these people who have been working on construction projects of this kind might happen to come our way, and I am sure in this case I might have thought of Garre's disease. In one of these cases the lateral view of the tibia would have made me think of the sclerosing type of osteitis. In both shoulders I noted an absorption of the neck with the production of what would correspond to a coxa vara of the hip. I am amply repaid for staying over to see these pictures and hear Doctor Coley.

DR. BRADLEY L. COLEY (New York, N. Y., in closing): In answer to Doctor Cohn's question, I would say that I think the bony deformity he mentioned in the upper humerus is also present, to a certain extent, in the views of both hips of the first patient. It is apparently due to a gradual softening during the period when the infarcted area of bone is becoming calcified.

It seems to me that with conditions as unsettled as they are to-day, and with submarine and other types of warfare so general, it is not beyond the realm of possibility that some of us may encounter more of these cases as time goes on, particularly if—as it now seems most unlikely—this country eventually becomes involved in another World War.

A MODIFIED SPUR-CRUSHING CLAMP AND ITS USE*

CHARLES H. WATT, M.D.

THOMASVILLE, GA.

IT IS generally agreed that the exteriorization type of operation in some form, for resection of the left colon and upper sigmoid, carries with it the minimum of risk. The procedure commonly known as the Mikulicz multiple-stage resection has a rather limited field but in a modified form permits just as radical removal as any other operation. Exteriorization resections, such as that described by Rankin[3] and known as the "obstructive resection," have greatly extended the field of this procedure without increasing the risk to any appreciable extent. However, there are several objectionable features, acquired at the price of safety, which, if reduced to a minimum, should give still broader scope to this excellent procedure.

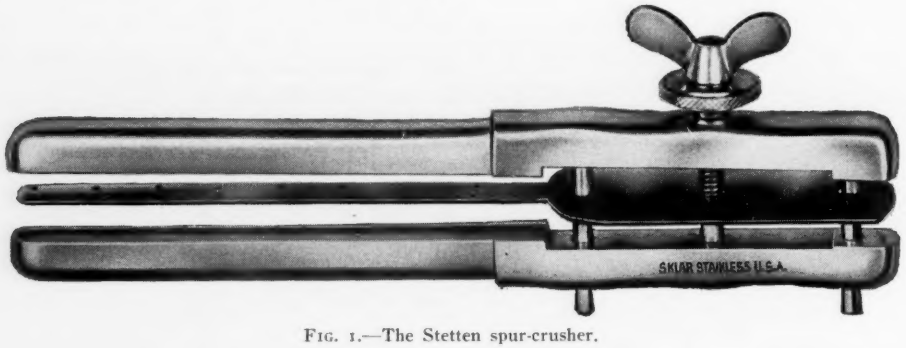

FIG. 1.—The Stetten spur-crusher.

It is not the purpose of this presentation to enter into a discussion of the advantages or disadvantages of any operation but to call your attention to the writer's modification of a spur-crushing clamp which, for him at least, has eliminated some of the objectionable features of the exteriorization type of resection referred to above.

These objectionable features may be briefly listed as: (1) Long convalescence due to multiple stages; (2) a colostomy with its accompanying disagreeable features; (3) repeated applications of the spur-crushing clamp often associated with pain, nausea and vomiting; (4) the risk of clamping beyond the desired area and the associated danger of injury to small bowel; and (5) the difficulty in closing the colostomy in a small percentage of cases.

The writer recently directed a brief questionnaire to a small group of surgeons known to be especially interested in surgery of the colon. The object of this questionnaire was to find out whether or not these men, in their extensive experience, had encountered the same difficulties the writer had

* Read before the Fifty-second Annual Meeting of the Southern Surgical Association, Augusta, Ga., December 5, 6, 7, 1939.

with the exteriorization operation, in his more limited experience. There was a certain degree of satisfaction obtained in learning that they, too, found it necessary, as a rule, to make at least two, and often three, applications of the spur clamp, and that in a certain percentage of their cases the colostomy failed to heal completely after the first attempt at closure. All were of the opinion that such failure is usually due to insufficient destruction of the spur.

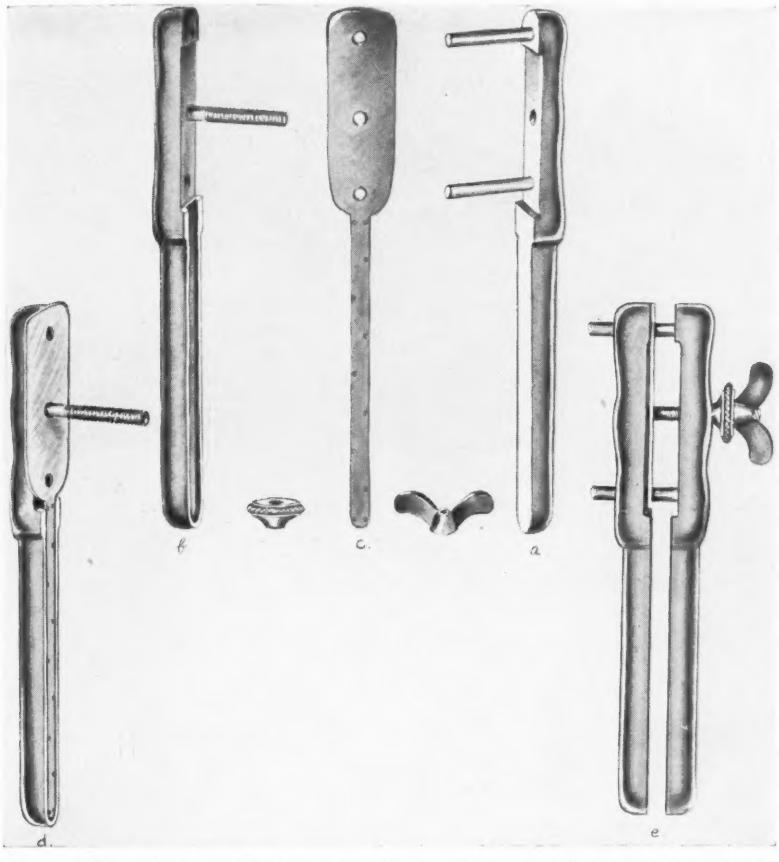

FIG. 2.—The author's modification of the Stetten clamp. Detailed description of this clamp will be found in the text.

This repeated application of the clamp, with its accompanying discomfort, which may be followed by failure of the colostomy to heal after operative closure, is likely to be very disheartening to an already discouraged patient.

Any clamp or improvement in technic that will permit one to obtain a sufficient cut through the septum with one application and at the same time insure the safety of the adjacent small bowel will reduce both the discomfort and the hazards of the operation and shorten the period of convalescence.

In performing the exteriorization operation, most surgeons prefer to suture the two ends of the severed bowel together for a distance of four or five inches to lessen the chance of clamping adjacent small bowel. However,

as pointed out by Jackson[1] before this Association last year, a five-inch suture line on the bowel may shrink to less than three within a short while. An attempt, therefore, to cut the spur to the approximate depth of the area sutured may result in the jaws of the clamp biting beyond this area and injuring adjacent bowel.

Edema and induration at the site of the resection usually make it diffi-

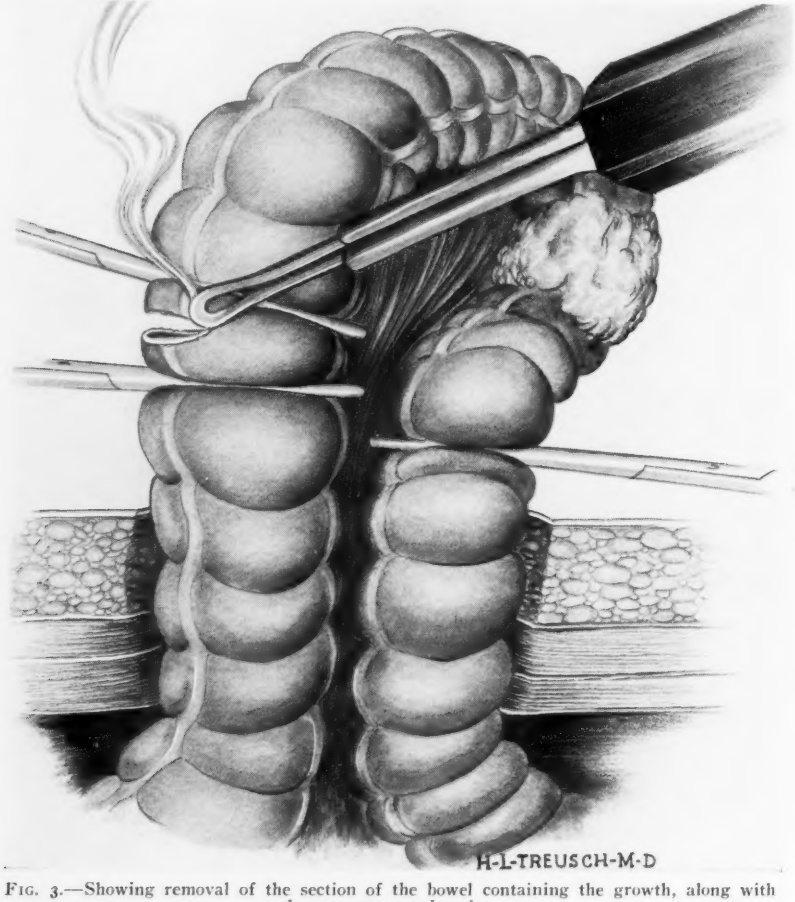

FIG. 3.—Showing removal of the section of the bowel containing the growth, along with the mesentery and nodes.

cult to apply a clamp for any depth until such reaction has subsided. Because of this some surgeons prefer to wait several weeks before attempting to cut the septum. This means a delay in the ultimate closure of the colostomy and prolongs convalescence.

The clamp the writer wishes to call to your attention permits the operator to accurately determine the location and depth of the opening to be made in the septum with one application and insures against injury to adjacent bowel. The crushing force may be applied the moment the obstructing clamps are removed or at any time thereafter.

Description of Clamp.—The clamp is a modification of the Stetten spur-crusher (Fig. 1). This clamp is in common use to-day and need not be described. One advantage of this style clamp is that it produces equal pressure throughout the entire crushing surface at all times.

In the writer's modification of this clamp the jaws are slightly wider and are four inches long (Fig. 2). Instead of grooves on the crushing surface,

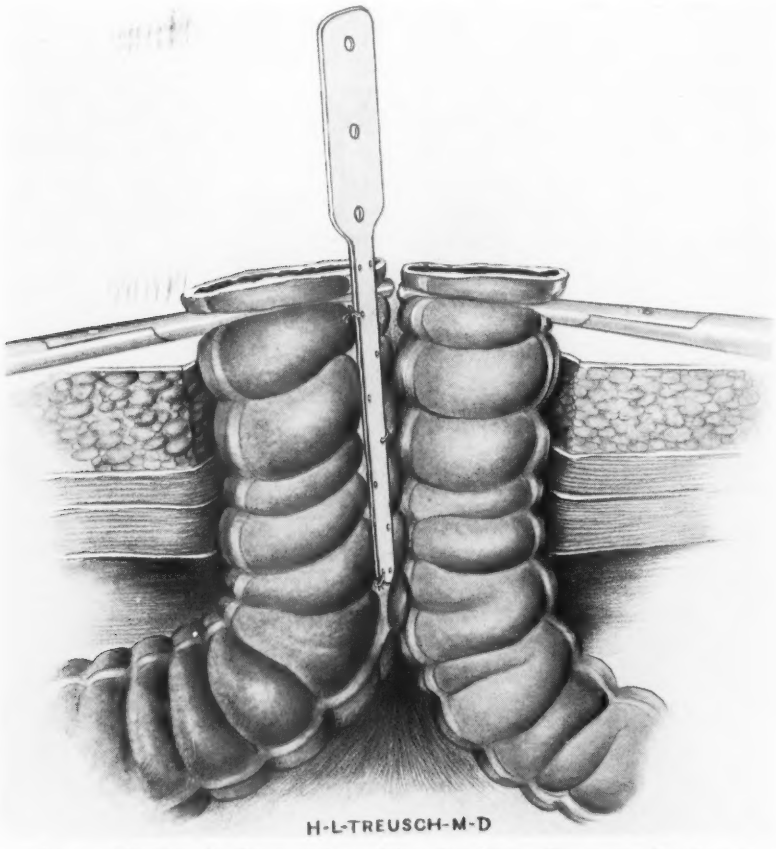

FIG. 4.—Showing the first step in applying the clamp. The tongue has been anchored to the longitudinal band of one end of the severed bowel by three silk sutures. Note that these are not all placed on the same side of the tongue, thus preventing rotation. The mesentery has been rotated laterally.

one jaw, called the upper jaw (Fig. 2 a) for identification purposes, is perfectly smooth, while the other, called the lower jaw (Fig. 2 b), is hollowed out to receive the third part, called the tongue (Fig. 2 c), leaving a narrow crushing surface, or rim, around the groove.

This third part, or tongue, rather resembles a spatula with a blade and handle. The blade is made so that it fits loosely in the groove in the lower jaw (Fig. 2 d), and staggered every half inch along its edges are small perforations or suture holes. The handle contains three holes that fit over the posts on the jaw handles when the clamp is assembled (Fig. 2 e).

Method of Using Clamp.—The portion of bowel containing the area to be resected is delivered and excised, along with its mesentery and nodes, between crushing clamps (Fig. 3). The two ends of the cut bowel are then brought together in the usual way for a distance of three or four inches, if possible. But, instead of suturing these together at this point as is usually done, the tongue part of the clamp is sutured to one of the bowel ends, prefer-

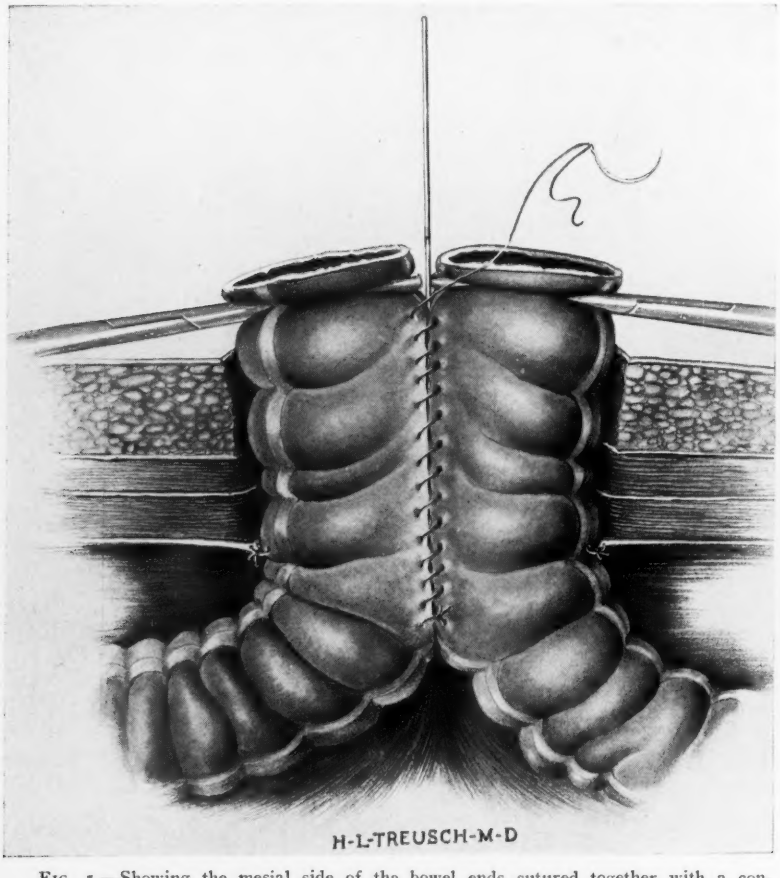

FIG. 5.—Showing the mesial side of the bowel ends sutured together with a continuous suture of fine chromic catgut—burying the blade of the tongue like a finger in a glove. An atraumatic needle is usually used instead of the large eye shown here by the artist. The peritoneum is shown stitched to the bowel.

ably along a longitudinal band, by three or four interrupted, fine silk sutures (Fig. 4). This blade is so placed that its tip will be at the base of the future septum or spur. After the blade of the tongue has been anchored in this manner, the two ends of the bowel are sutured together (Fig. 5) in the usual way, with a continuous suture of fine chromic catgut, so that the blade is buried between the bowel walls like a finger in a glove. If the bowel has been rotated mesially, as suggested by McNeely and Lichtenstein,[2] with the mesentery laterally, it is only necessary to suture the bowel ends together on the mesial side of the blade and around the tip. The remaining steps in

this stage of the operation consist in the usual suturing of the peritoneum around the two bowel ends and loosely closing the wound edges, leaving the bowel ends, occluded by the crushing clamps, protruding from the wound with the handle of the tongue rising between them like a periscope.

At the end of 48 or 72 hours, depending upon the patient's condition and comfort, both obstructing clamps are removed and the jaws of the crushing

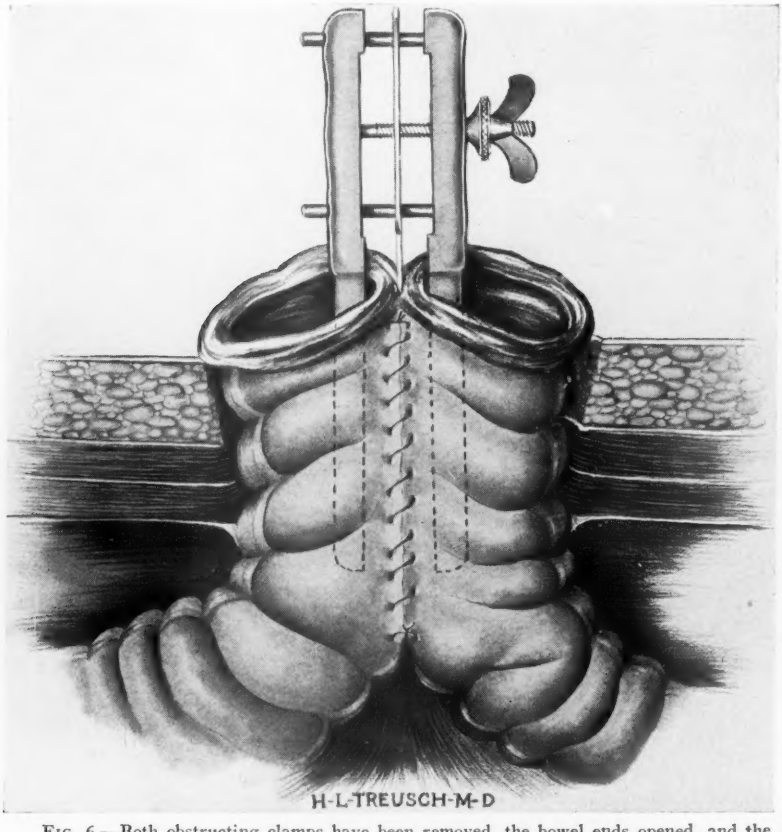

FIG. 6.—Both obstructing clamps have been removed, the bowel ends opened, and the jaws of the crushing clamp have been fitted to the blade ready for the crushing force to be applied by screwing down the wing-nut.

clamp are applied at once, one in the ascending and the other in the descending arm of the double-barrel colostomy (Fig. 6). It is not necessary, as a rule, to feel with the finger within the bowel ends for the spur before inserting the jaws. The tongue or guide shows the way and has kept the bowel "ironed out." When the posts of the jaws fit in the holes on the tongue handle there can be no question as to where the jaws are. One may be sure: (1) That no stray loop of bowel has gotten in between the jaws of the crushing clamp; (2) that the septum will maintain its original length, because it has been kept "ironed out" by the tongue blade; and (3) that one application of the clamp will crush the spur as far down as the tongue blade is sutured, namely, to the base of the spur.

As soon as the clamp is fitted, pressure is exerted by screwing down on the wing-nut. This is done several times each day and is usually painless. The entire clamp with the necrotic portion from the septum usually comes away on the sixth or seventh day. By this time the edema of the bowel ends has greatly subsided and one may attempt closure of the colostomy at this time with a reasonable assurance of its remaining closed. However, the removal

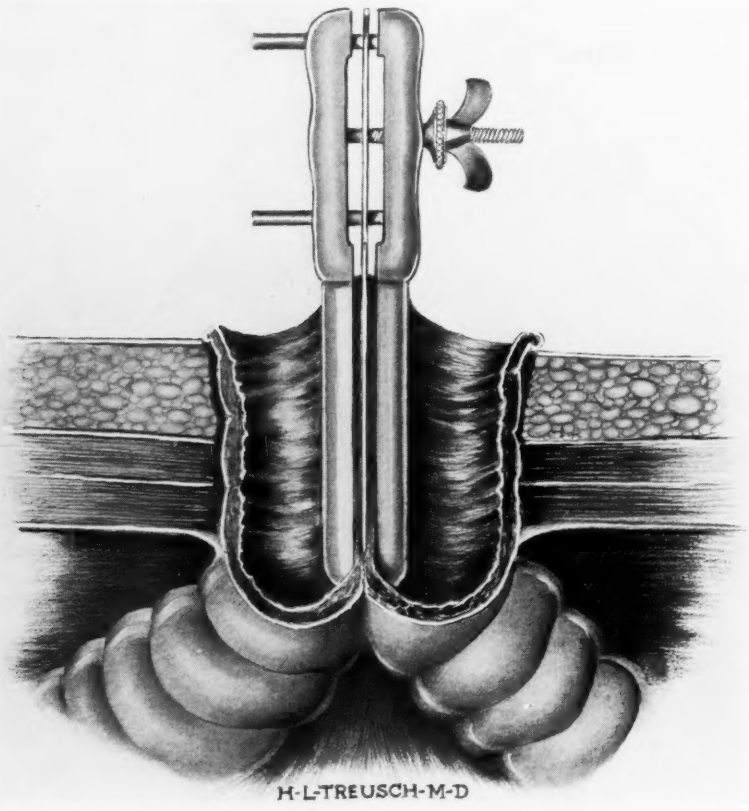

FIG. 7.—The mesial sides of the bowel ends have been removed by the artist to show the relationship between the bowel walls and the different units of the clamp.

of the clamp is usually the beginning of a through passage of bowel contents soon to be followed by normal bowel movements and lessening of the colostomy drainage. Since both of these are encouraging to the patients, we usually allow them to leave the hospital after ten days or two weeks.

Some of these cases would no doubt heal without further surgery, but unless there is some contraindication, we prefer to have them return after two weeks for an extraperitoneal closure, in order to eliminate a possible hernia. With a deep, wide opening in the septum, flush with the bowel floor, the closure of the colostomy transversely is relatively simple and should be followed by very few failures.

Two theoretic objections have been raised to the employment of this tongue,

namely: (1) The additional sutures increase the risk of infection; and (2) the blade tends to allow drainage from the bowel to seep down between the approximated surfaces.

Naturally, care should be exerted in introducing all sutures into the bowel wall. The sutures anchoring the tongue are shut off from the general peritoneal cavity by the running suture commonly used, and by applying the clamp as soon as the obstructing clamps are removed there is no chance for the bowel contents to enter the pocket containing the tongue-blade.

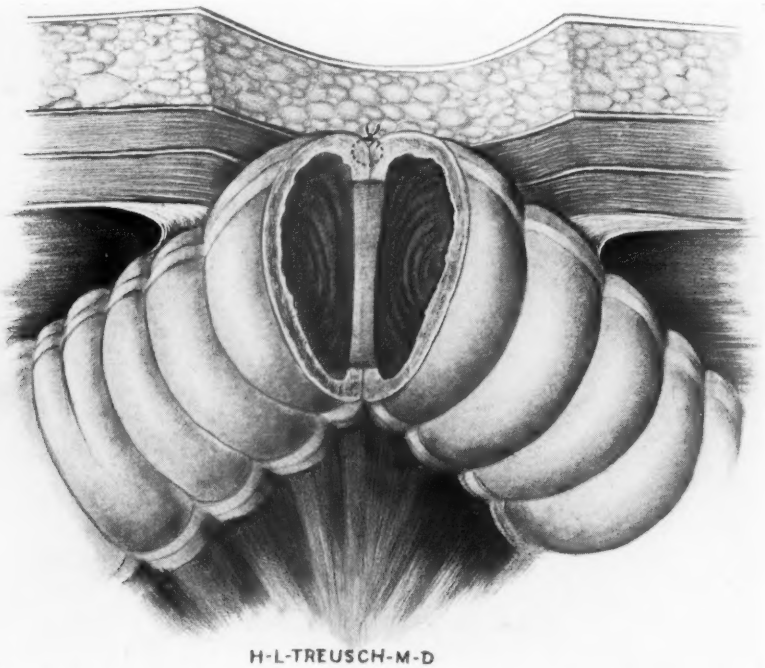

FIG. 8.—Extraperitoneal closure of the colostomy. Suture line transversely.

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BOOKS RECEIVED

THE 1939 YEAR BOOK OF NEUROLOGY, PSYCHIATRY AND ENDOCRINOLOGY. Edited by Hans H. Reese, M.D., Nolan D. C. Lewis, M.D., and Elmer L. Severinghaus, M.D. GENERAL THERAPEUTICS. Edited by Bernard Fautus, M.S., M.D., and Leroy H. Sloan, M.D. DERMATOLOGY AND SYPHILOLOGY. Edited by Fred Wise, M.D., and Marion B. Sulzberger, M.D. UROLOGY. Edited by John H. Cunningham, M.D. PEDIATRICS. Edited by Isaac A. Abt, D.Sc., M.D., and Arthur F. Abt, M.D. OBSTETRICS AND GYNECOLOGY. Edited by Joseph B. DeLee, M.D. and J. P. Greenhill, M.D. Chicago: The Year Book Publishers, Inc., 1939.

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SHALL WE LIVE ON. By W. A. Newman Dorland, M.D. New York: Fortuny's, 1940.

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